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**Mood Manipulation and Attentional Processes:  
Electrophysiological Investigations of the  
Affect-Cognition Interaction**

**by**

**Gaynor Evans**

A thesis submitted in partial fulfilment of the requirements for the degree

of

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I would like to dedicate this thesis to the memory of my father.



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## **Declaration**

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I, Gaynor Evans, declare that the work carried out for this thesis was the author's own. I confirm that this thesis has not been submitted for a degree at another University.

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## Summary

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Although there has been considerable research into the effects of major affective disorders on perception and cognition, there has been less focus on the influence of everyday fluctuations in mood on general cognitive skills. Neurocognitive models of affect-cognition interactions implicate frontal cortical networks and predict that where task control is reliant on such networks, there will be a greater negative impact of mood change.

The initial study in this thesis compared the effectiveness of 3 standard mood induction techniques as assessed by a subjective mood assessment instrument. The most effective changes were only evident with the induction of negative mood, using the Velten Mood induction technique, which was therefore adopted for subsequent studies.

Three further studies employed a within-subject design, investigating the effect of neutral and negative mood on 3 tasks selected for a) increasing levels of cognitive demand and complexity, and b) the increasing involvement of frontal areas of control. Event-related potentials (ERPs) were measured using a 128 channel dense array system. It was predicted that mood induction would differentially activate the frontal areas, as measured by increased negative amplitude in frontal ERPs, lateralised to the left hemisphere, and be associated with changes in task performance and the associated ERP 'signature'.

The first task, an 'odd-ball' task, was of low cognitive demand and associated with central-parietal control, and showed no disruption at the behavioural or cortical level. The second task, a standard Stroop task, also showed no behavioural disruption but there were mood-related differences in frontal ERPs. Increases in negativity caused the pattern of activity associated with congruent and incongruent trials to be reversed. The final task, an N-back working memory task, again showed minimal disruption at the behavioural level, but significant differences in lateralised frontal activity as a function of mood. Again, increased negativity within in left hemisphere led to a reversal of asymmetry during the cognitively demanding 3-back task.

The data are interpreted in terms of Ellis and Ashbrook's Resource Allocation Model, which predicts that depressed moods lead to a reduction in the capacity of resources allocated to the control of cognitive tasks. It is concluded that the maintenance of performance is associated with increased allocation of cortical resources.

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# Chapter 1

## Affective Neuroscience – Mood and cognition

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### 1.1 The Development of Affective Neuroscience

Understanding the way in which affect influences cognitive performance has always been of great philosophical interest (Forgas, 1995) however scientific enquiry under the umbrella of *Affective Neuroscience* is still a relatively new branch of psychology. By adopting an inter-disciplinary approach, the last thirty years have seen a growth in our understanding of the way in which affect interacts with cognition (Ellis, 1991). The influence of affect can be seen at all levels of information processing, through the search for and retrieval of information, to the attentional processes directed at that information, and finally the way in which that information is processed and interpreted (Au, Chan, Wang, & Vertinsky, 2004).

A considerable amount of research has been conducted upon the phenomena of ‘cognitive bias’ in anxiety and depression, where the valency of mood influences perception (see for example (Eysenck, MacLeod, & Mathews, 1987; Sutton & Davidson, 2000)). However this thesis is concerned with the influence of mood upon cognitive processes. Using induced mood states, affect has consistently been shown to influence a number of cognitive processes, including memory (e.g. Ellis, Thomas, McFarland, & Lane, 1985; Pereg & Mikulincer, 2004; Murray, Whitehouse, & Alloy, 1999), risk preferences (e.g. (Mann, 1992), decision making (e.g. Yuen & Lee, 2003), problem solving (e.g. Isen, 1983; Isen, 1984; Isen & Daubman, 1984; Isen, Daubman, & Nowicki, 1987), deductive reasoning (e.g.

Oaksford, Morris, Grainger, & Williams, 1996), text comprehension (e.g. Ellis, Ottaway, Varner, Becker, & Moore, 1997), and judgments (e.g. Forgas, 2002; Isen & Means, 1983; Arkes, Herren, & Isen, 1988; Isen, Means, Patrick, & Nowicki, 1982). Furthermore, similar patterns of cognitive influences are seen in clinical patients suffering from depression, including memory (e.g. Elwart, Rinck, & Becker, 2003; Potts, Camp, & Sturcke, 1989), and decision making (Murphy et al., 2001; Okwumbua, Wong, & Duryea, 2003).

## **1.2 The Basis of Affect-Cognition Interactions**

Theoretical descriptions of these interactions traditionally fall into two mutually exclusive hypotheses, broadly referred to as facilitation and suppression (Oaksford et al., 1996). The basis for both these approaches is the way in which affect influences the central executive, one of the triad of components within working memory (Oaksford et al., 1996). The central executive plays an important role within information processes as it acts as the cognitive workspace where information is manipulated in light of input from the working memory sub-systems, the articulatory loop and visuospatial sketch pad (Baddeley, 1986; Baddeley, 2000).

### **1.2.1 The Facilitation Approach**

Oaksford et al (1996) proposed this hypothesis as an extension the work on mood and creative problem solving by Alice Isen and her colleagues (Isen, 1983; Isen, 1984; Isen, 1987; Isen et al., 1984; Isen et al., 1987). In one investigation into the influence of affect on cognition, Isen et al. (1987) employed the Dunker (1945) candle problem. In this task, participant must decide how they can support a lighted candle on a door upon being presented with a box of tacks, some matches and a candle. The solution of this problem

involves the realisation that the box of tacks has multiple uses, and therefore can be used as a candle-holder attached to the door by the tacks themselves (Dunker, 1945).

Using film as an induction procedure, the investigation compared both positive and negative mood with 2 control conditions (neutral film and no film) (Isen et al., 1987). Performance on the task was significantly better for the positive affect group only. Isen et al (1987) felt the facilitation of performance was through two avenues, long term memory and information processing. It was believed that the induced positive mood acted as a retrieval cue for positive material held in long term memory. As positive memories are more likely to be associated with everyday occurrences, it was concluded that more functions for the objects would be prompted by the influx of positive memories (Isen et al., 1987).

In addition, Isen et al (1987) believed that the positive mood would act directly upon the information processing involved during problem solving, rather than influencing the resources available to those processes. Using a similar approach, Oaksford et al (1996) concluded a similar influence of positive affect may be seen on the more analytical task of deductive reasoning.

### **1.2.2 The Suppression Approach**

Of the two approaches, the view that affect suppresses cognitive performance has received more experimental investigation. The theoretical basis for this approach came from the assumption that conscious attention was reliant upon a fixed capacity of cognitive resources (Kahneman, 1973). Taken together with the postulation that a distinction exists between automatic and effortful processes (Hasher & Zacks, 1979), the resource allocation model

(RAM; Ellis & Ashbrook, 1988) was proposed to explain the interaction between mood and memory.

The essence of the RAM is that the cognitive capacity which can be allocated to a criterion task is regulated by emotional states, through the phenomenon of mood congruent memory (Ellis et al., 1985; Ellis, 1991). When in a depressive or sad mood the production of irrelevant thoughts increases (Ellis et al., 1997), due to affectively compatible information being selectively processed at the expense of affectively unrelated information (Blaney, 1986).

Ellis & Ashbrook (1988) argued that emotional states could influence resource allocation during cognitive processing in two ways. A segment of the total capacity could be 'preempt' by the depressive or sad mood, leaving sufficient resources to satisfactorily process the criterion task. Alternatively the resources available to the criterion task could be quantitatively reduced by the depressed mood (Ellis et al., 1988; p27). This highlights the most significant aspect of Ellis & Ashbrook's (1988) model, namely it is the resource capacity allocated to a criterion task which is important, rather than what resources are available (Ellis, 1991).

Therefore RAM states that during a depressed or sad mood an individual will experience an increased number of irrelevant thoughts whilst they are completing a criterion task (Ellis, 1991). These distracting thoughts can be described as 'extra-task' processing, such as focusing upon the depressive state being experienced (Kihlstrom, 1989; Oaksford et al., 1996), or 'task-irrelevant' processing of task features such as the font used (Kihlstrom, 1989; Ellis, 1991). As the processing of irrelevant thoughts increases, there is a reduction in

the resource capacity allocated to criterion task which leads to a performance deficit (Ellis, 1991). Support for the resource allocation model comes from both clinical and healthy participant populations.

### **1.3 Assessing the Resource Allocation Model**

As previously stated, it is the central executive component of working memory which is thought to be the location of the affect-cognition interaction (Oaksford et al., 1996). A classic way of testing central executive functioning is the Tower of London task (Shallice, 1982), which comprises of three pegs of differing heights upon which three incrementally sized disks can be placed. The aim of the task is to move the disks from an initial pattern to a goal state adhering to a series of rules, in the minimum number of moves possible (Shallice 1982). Using this task, Watts, MacLeod & Morris (1988) showed that task-irrelevant processing in clinically depressed patients lead to a performance deficit, therefore providing support for both the model and its fundamental premise.

Further evidence for the detrimental consequences of mood congruent thoughts came from Ellis, Seibert, & Herbert (1990). Not only were they able to demonstrate that an induced negative mood increased the production of negative self thoughts, participants in this mood state also exhibited poorer performance upon a recall task (Ellis et al 1990). Seibert & Ellis (1991) showed that participants experiencing induced happy and sad moods reported more irrelevant thoughts during a criterion task, both during and following the criterion memory task (Ellis et al., 1997). Furthermore, they reported a strong correlation between the proportion of irrelevant thoughts and recall performance (Seibert & Ellis, 1991).

It is apparent that the influences of affect proposed by the facilitation and suppression hypotheses oppose one another. In their study of deductive reasoning, Oaksford et al (1996) directly compared these two models of cognitive performance. This study employed a modified, deontic version of Wason's (Wason, 1966; Wason, 1968) selection task thereby removing the floor and ceiling effects associated with the original version.

The theme of the task was based upon the Cheng & Holyoak (1995) immigration task (Oaksford et al., 1996), where participants are instructed that they are immigration officials who were responsible for checking entrance documents for inoculation information. In particular, they are concerned with whether 'Form H' conforms to the rule: 'if a passenger's form says "ENTERING" on one side, then the other side must include cholera' (Oaksford et al., 1996). Correct performance plateaus at 60% when participants are given no explanation as to why this disease check is necessary (Cheng & Holyoak, 1985), therefore this condition was chosen so that potential facilitation or suppression effects could be observed (Oaksford et al., 1996).

Using film to induce positive and negative moods, Oaksford et al (1996) demonstrated that transient mood states, both positive and negative lead to a deficit in reasoning performance. Furthermore, similar results were seen when participants performed the deontic reasoning task with a concurrent monitoring task. Oaksford et al (1996) concluded that both the transient moods and the monitoring task load cognitive functioning leading to performance suppression, in line with the resource allocation model.

In their third study, Oaksford et al (1996) wished to establish whether the reasoning deficit witnessed in their earlier studies was a result of direct or indirect suppression. Again, the



Tower of London task was chosen due to its reliance upon executive functioning. For the task to be completed successfully, the participant must create and appraise internalised action plans within the cognitive workspace that is assumed by many to be provided by our working memory (Oaksford et al 1996). In relation to planning time, no significant differences were found between the positive, negative and neutral conditions, however the number of moves executed was sensitive to the participant's mood state (Oaksford et al 1996). Those in a positive mood took significantly more moves to rearrange the disks from the initial to the goal state than both the neutral and negative mood groups. Surprisingly, the same effect was not associated with the negative mood state, where there were no significant differences between the number of moves taken to complete the task (Oaksford et al 1996).

Consequently, they felt able to provide confident conclusions for the influence of positive mood on cognitive processes alone. As only the number of moves required to complete the task differed from the control group, it was concluded that individuals in a positive mood were able to respond to time pressure in a consistent manner but their performance deficit was the result of not being able to construct efficient action plans (Oaksford et al 1996).

Although the result of the final study prevented them from making reliable conclusions on the performance deficits seen during negative moods, the earlier results coupled with the trends witnessed allowed them to make tentative allusions. During the Tower of London task there was a tendency for the individuals in a depressed mood state to spend more time planning their attempt to move from the initial to goal pattern. Therefore it is possible that in order to compensate for the depleted resources allocated to the task, the individuals take longer to generate and assess their strategies (Oaksford et al 1996).

Other researchers have focused upon trying to establish the processes which may mediate the phenomenon of mood congruent memories. As previously stated, Blaney (1986) described mood congruent memories as being the result of the selective processing of information analogous to the dominant emotional state. Although there is inconsistency within the literature, definitions of emotional state describe a multi-dimensional condition which includes physiological and cognitive arousal (see for example, Mayer & Salovey, 1988; Schachter & Singer, 1962)). Consequently the activation these distinct systems creates two mechanisms which may be responsible for the mood congruence effects underlying the resource allocation model (Varner & Ellis, 1998).

Physiological arousal is associated with a number of salient visceral experiences, for example raised heart rate and sweating. Therefore it would be expected that selective processing of affective information would be as a result of the congruence between the individual's current physiological state and that elicited by the stimulus (Varner et al., 1998). Activation of cognitive processes, however would lead to a reduction in cognitive resources being allocated to the criterion task, as outlined in Ellis & Ashbrook's (1988) resource allocation model (Varner et al., 1998).

In order to isolate the mechanism responsible, participants' performance upon a word recall task was compared using three induction procedures: mood, schema and arousal (Varner et al., 1998). The mood induction procedure resulted in two experimental conditions, a depressed mood which would be associated with both physiological and cognitive arousal and a neutral mood which would result in irrelevant cognitive activation and no physiological arousal. Both mood states were generated by the participant reading either a series of negatively charged or neutral statements (Velten, 1968).

A schema induction paradigm was used to produce the experimental condition in which the individual would experience cognitive activation without any physiological arousal. This was achieved by the participant reading a series of statements which described the activities involved in writing an academic paper. Finally, physiological arousal was attained by the participant repeatedly walking on and off a step. Following the induction procedure the participant attempted to learn a word list containing a mix of words with a negative valence and those described as organisational, which were related to writing a paper (Varner et al., 1998).

The study clearly demonstrated that the cognitive activity associated with the experience of emotional states is responsible for mood congruent recall. Participants experiencing a depressive mood state showed selective recall of negative words over organisational words while those who received the schema induction exhibited selective recall of organisational words over negative words. Finally the individuals who were physiologically aroused showed no categorical bias in the words recalled (Varner et al., 1998). Furthermore, a similar pattern of recall bias was seen when the participants underwent the induction procedures in between learning and recalling the words (Varner et al., 1998). Therefore it was concluded that cognitive and physiological processes activated during the experience of an emotional state do not interact during mood congruent recall. The phenomenon of mood congruent recall is mediated by cognitive processes alone (Varner et al., 1998).

As well as providing support for the Resource Allocation Model, this study highlights the proposition that the way in which thoughts will have an impact upon cognitive processing is dependent upon their relationship to the information contained within the criterion task (Ellis et al., 1988; Ellis, Varner, & Becker, 1993). Therefore if an individual's thoughts are

irrelevant to the criterion task this will inhibit cognitive processing, whereas if they are relevant to the information associated with the criterion task then processing will be facilitated (Varner et al., 1998).

Interestingly, this aspect of the resource allocation model seems to have been largely ignored by researchers. Take for example Oaksford et al (1996) who described how the findings of affect-cognition research fall into facilitation versus suppression debate, and cite the work of Isen and her colleagues as supporting the facilitation hypothesis (for example, Isen et al., 1987)). However, it should be remembered that Isen et al (1987) concluded that the improvement in creative cognitive performance demonstrated in their study was a result of the positive affect increasing positive memories. Therefore, rather than being in opposition of the Resource Allocation Model, the work of Isen and her colleagues supports the less familiar proposal that task-relevant thoughts will facilitate cognitive performance (Varner et al., 1998).

This comprehensive understanding of the Resource Allocation Model aligns itself with the approach taken by Joseph Forgas in understanding the way in which affect influences social judgment (see for example, Forgas, 1995). Forgas (1995) describes the process of affect infusion which postulates that “affectively loaded information exerts an influence on and becomes incorporated into the judgemental process” (Forgas, 1995, p39). The most important aspect of this Affect Infusion Model is the aim to be all encompassing, therefore not only providing explanations of situations where affect influences judgement but also those where no influence is exerted, and situations where the judgement given is in contrast with the individuals affective state (Forgas, 1995). Therefore, although the Resource

Allocation Model arose out of a cognitive description of depression it should be seen as an inclusive explanation of the influences of affect on cognition.

Another common misconception is that should a study show no interaction between affect and cognition, then this is viewed as being evidence against the model under investigation. In fact, Ellis (1991) explicitly states that there is no empirical evidence to assume there will be affect-driven deficits seen in all aspects of cognitive performance, and fully supports explorations of the conditions under which such interactions occur. In particular, the attentional framework of the Resource Allocation Model gives rise to the proposition that in situations where a depressed individual has their attention fixed upon the criterion task a reduction or elimination of deficit will occur (Ellis et al., 1988).

The influence of attention on the affect-cognition interaction was elegantly investigated in a study by Hertel & Rude (1991). Although the Resource Allocation Model provides an apt explanation as to why depression leads to a cognitive performance deficit, there is another viable explanation, namely motivation (Ellis et al., 1997). It is possible that what is seen in these experiments is the depressive state causing the participant to be less motivated to perform the task (Ellis et al., 1997). Therefore there would be adequate cognitive resources applied to the criterion task, but the participants would have insufficient initiative to complete the task successfully (Hertel & Rude, 1991). Hertel & Rude (1991) manipulated the attentional focus within an incidental learning task which had been employed in a number of studies investigating the influence of affect upon cognition (for example, Ellis, Thomas, & Rodriguex, 1984). The basic structure of the experiment is that participants are presented with a target word followed by the sentence, their aim is to judge whether the target word would fit into the sentence. Attentional focus was increased by presenting the

target word for a brief period of time prior to the sentence. The participants would then have to repeat the target word and verbally indicate whether the target word would fit the sentence (Hertel et al., 1991). During the unfocused condition the target word remained on the screen throughout the sentence presentation, no repetition of the target word was required and their suitability judgement could be made at any time (Hertel et al., 1991).

When depressed patients and healthy individuals were compared, clear attentional differences were seen. Where the focus of attention was restrained by the characteristics of the task, no depressed deficits were seen. However, when variations in attention were possible the conventional performance deficits were associated with the depressed individuals (Hertel et al., 1991). This does not however, automatically act as support of the motivational interpretation as the Resource Allocation model does predict the weakening of performance deficits when attentional resources are more focused upon the criterion task (Ellis et al., 1988).

Ellis et al (1997) addressed the issue of motivation further in an investigation of the influence of affect on a comprehension task. It was demonstrated that participants in a depressed mood state were inferior at identifying contradictions within a prose passage, regardless of whether they were informed of the presence of errors. In addition, those individuals who were more motivated displayed a higher number of false identifications which is inconsistent with a purely motivational explanation (Ellis et al., 1997).

Kihlstrom (1989) suggested that another way of addressing the possible influence of motivational factors would be to compare the influence of positive and negative moods on cognitive processing. As positive moods are invigorating, a motivational account would

predict that performance deficits would be specific to depressed and sad moods. However, if both positive and negative moods lead to task-irrelevant processing then deficits would be associated with both mood states in line with Ellis & Ashbrook's (1998) model (Kihlstrom, 1989). Again, the Resource Allocation Model receives endorsement from the literature with both positive and negative influenced deficits seen in free recall tasks (Seibert et al., 1991; Ellis, Seibert, & Varner, 1995) and deontic reasoning (Oaksford et al., 1996).

Varner & Ellis (1998) discuss further the conditions under which the Resource Allocation Model proposes that mood congruence memories will interfere with a criterion task. First the items which are being processed should contain at least two discrete categories of information, thus creating a situation where one category can be selected at the expense of the other (Varner et al., 1998). Secondly, the thoughts should have a dichotomous relationship with the categories of information contained within the material to be processed. Therefore they should be relevant to the processing of one category of information and irrelevant to the processing of the second category (Varner et al., 1998).

The findings of Varner & Ellis (1998) can be seen to support this proposition; both conditions are met within the depressed and organisational conditions while the arousal and neutral mood conditions do not satisfy the criteria. Therefore during the depressed condition the participant's attention was focussed upon their sad mood which was related to the negative words but unrelated to the organisational words. Likewise, in the organisational condition, the participant's effort was placed upon the writing of a paper therefore creating an association with the organisational words but not the negative words (Varner et al., 1998).

It is apparent that the majority of experiments within this area concentrate upon the effects of depressed or sad moods upon cognitive performance. One of the main reasons for this is the relative difficulty in successfully inducing a positive mood in a laboratory environment. However, more salient to the resource allocation model is the neurophysiological relationship between the location of affect-cognition interactions and depressive episodes.

#### **1.4 The Neurological Correlates of Depression**

As with many psychopathological disorders, the aetiology of depression has been found to be a complex mix of genetic, environmental, developmental and neurological factors (Soares & Mann, 1997). The situation is complicated further by evidence that the neurological factor can be in the form of neuroendocrine, neurochemical, or neurophysiological abnormalities (Drevets & Todd, 1997). Regardless of their origin, these neurological factors contribute to the development of depression in one of two ways, either abnormalities that cause the circuitry involved in the expression of emotions to malfunction and/or abnormalities that render the individual susceptible to psychosocial stressors (Soares et al., 1997).

The cyclic nature of depression and the lack of a substantial relationship with indices of generalised anatomical damage have led to the assumption that these abnormalities affect a distributed modulatory system rather than causing permanent changes in connectivity and neurotransmission (Goodwin, 1997; Soares et al., 1997). Coupled with evidence showing secondary depression can occur following lesions and anatomical correlates of primary depression, this modulatory system is widely postulated to be based upon a frontal-subcortical circuit (Figure 1.1; Robinson, Kubos, Starr, Rao, & Price, 1984; Buchsbaum,



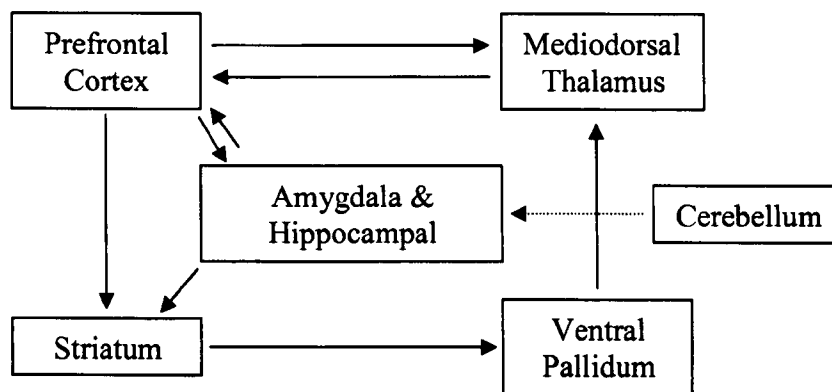


Figure 1.1 The neuroanatomical model of mood regulation (adapted from Soares & Mann 1997).

1986; Jeste, Lohr, & Goodwin, 1988; Nasrallah, Coffman, & Olson, 1989; Starkstein & Robinson, 1989; Beats, 1991; Drevets et al., 1992; McDonald & Krishnan, 1992; Cummings, 1993; George, Ketter, & Post, 1993; Guze & Gitlin, 1994; Mayberg, 1994; Mega & Cummings, 1994; Austin & Mitchel, 1995; Soares et al., 1997). Evidence from lesion, neuroimaging and induced mood studies have been cited in support of this hypothesis.

#### 1.4.1 Lesion Studies

Lesion studies provide a valuable insight into the anatomical correlates of various neurological disorders as they can provoke acute symptom (Goodwin, 1997). Whether caused by tumors, trauma or surgery, it appears that the frontal lobes are the critical area for lesions which give rise to mood disturbances (Cummings, 1995). In particular, it is thought that the circuitry described in Figure 1.1 is disconnected by the lesions (Soares et al., 1997). The strongest relationship between the development of secondary depression and lesions occurs when the lesion is caused by vascular abnormalities (Awad, Spetzler, Hodak, Awad, & Carey, 1986; Coffey, Figerl, Djang, Saunders, & Weiner, 1989; Lesser et al., 1991; Schmidt, Fazekas, Offenbacher, Lytwyn, & et al, 1991). This may account for the link

being unequivocal in elderly patients only (Soares et al., 1997), as cardiovascular disease is far more common in elderly patients.

Furthermore, the laterality of the lesion within the frontal lobe also has implications on the development of secondary depression. Left frontal lobe lesions are associated with higher incidence rates and an increased severity in the depression suffered (Lipsey, Robinson, Pearlson, Rao, & Price, 1983; Robinson et al., 1984; Robinson, Starr, Lipsey, Rao, & Price, 1984; Robinson, Boston, Starkstein, & Price, 1988; Eastwood, Rifat, Nobbs, & Ruderman, 1989; Astrom, Adolfsson, & Asplund, 1993; Herrman, Bartels, & Wallesch, 1993; Jorge et al., 1993). Where exceptions have occurred, there has been evidence of either subcortical damage (Starkstein, Robinson, & Price, 1987) or the lesion occurring in the posterior region of the frontal lobe (Folstein, Mailberger, & McHugh, 1977; Robinson et al., 1984). In general lesions occurring in the right frontal lobe are connected with the experience of manic episodes in bipolar depression (Cummings & Mendez, 1984; Forrest, 1982).

#### **1.4.2 Neuroimaging Studies**

The excellent spatial resolution of haemodynamic neuroimaging techniques such as Positron Emission Tomography (PET), Single Positron Emission Computerised Tomography (SPECT), and function Magnetic Resonance Imaging (fMRI) provide an unparalleled opportunity of investigating the neuroanatomical correlates of psychopathological disorders such as depression (Drevets, 2000). These techniques have demonstrated a constellation of cerebral blood flow and metabolism abnormalities in depressed individuals (Baxter, Gerner, Mazziotta, & Phelps, 1983; Baxter et al., 1985; Brodie et al., 1983; Gur et al., 1984; Gustafson, Risberg, & Silfverskiold, 1981; Mathew et

al., 1980; Phelps & Mazziotta, 1985; Silfverskiold, Gustafson, Johanson, & Risberg, 1979; Uytendhoef et al., 1983).

A common characteristic of both primary and secondary depression is abnormal region cerebral blood flow (rCBF) in the amygdala, anterior cingulate cortex and the frontal lobes (Drevets, 2001). However, the nature of the abnormality is less established as both reductions and increases in rCBF have been reported. A possible explanation for this inconsistency could be the sufferer's age, and consequently the type of depression presented. The reductions in blood flow are generally seen in elderly patients suffering from secondary depression (Bench et al., 1992; Curran et al., 1993a), while increases are seen in younger patients suffering from primary depression (Drevets & Raichle, 1992; Drevets et al., 1992). Therefore the reductions are thought to reflect a more global diminished mental state while the increases in perfusion index lead to the specific characteristics of depression, such as the dysphoric thoughts (Dolan et al., 1993).

As seen in lesion studies, a specific link to the frontal lobes has been discovered through neuroimaging studies. In particular, abnormal reductions in cerebral blood flow (RBF) have been found within the dorsolateral prefrontal cortex (DLPFC) (Drevets, 2000). Additionally, metabolism levels have been found to be decreased within the DLPLC when patients suffering a depressive episode during major depressive disorder are compared with those who are in remission (Bench et al., 1992; Biver et al., 1994; Mayberg et al., 1999). Furthermore, this area is thought to be specialised to the experience of emotion as the DLPFC does not appear to be activated during emotional processing (Drevets, 2000).

### **1.4.3 Mood Induction Studies**

Although the depth of emotion experienced during an induced mood will not reach the levels experienced during a depressive episode, there is evidence to show that the same areas of the brain are implicated during the experience of clinical depression and experimentally induced sadness (Drevets, 2000; Mayberg et al., 1999). Additionally, the nature of the abnormalities reported follows the same pattern as is seen in clinically depressed patients. The abnormal activation within the prefrontal cortex is associated with induced depressed moods in a number of studies, however it is unclear whether this abnormality represents a reduction in activity (Baker, Frith, & Dolan, 1997) or an increase in activity (Lane et al., 1997; Reiman, Lane, Ahern, & Schwartz, 1997).

### **1.5 The Neurological Correlates of Executive Functioning**

The frontal cortex, and in particular the dorsolateral prefrontal cortex (Brodmann areas 9 and 46 (Pandya & Yeterian, 1996), has been clearly implicated in depression. However the functionality of this region is not limited to the experience of emotional states, it is also plays an important role within cognitive abilities referred to as 'executive functions' (Petrides & Milner, 1982).

Executive functions can be thought of as a hierarchical system responsible for the control of action (Seitz, Stephan, & Binkofski, 2004). Through a series of sub-systems, such as volition, planning, selection, programming and the performance of movement, executive functions allow us to actively adapt to our environment (Seitz et al., 2004; Fuster, 2000). There is considerable neuroimaging evidence from a wide range of neuroimaging studies (for reviews see: D'Esposito et al., 1998a; Owen, 1997; Petrides, 2000b) that executive

function controlled tasks, such as working memory, are controlled by the dorsolateral prefrontal cortex (Petrides, 2000a).

## **1.6 Conclusions**

The Resource Allocation Model (Ellis et al., 1988) provides a fully integrated account of the influence of affect on cognitive performance. The essence of the Resource Allocation Model is that emotional states modulate the amount of capacity that is allocated to a criterion task through the elicitation of mood congruent memories (Ellis et al., 1988). The impact of these memories will be dependent upon the relationship between the information contained within them and that associated with the criterion task (Ellis et al., 1988; Ellis et al., 1993). Therefore task-relevant thoughts will lead to performance facilitation (for example, Isen et al., 1987) and task-irrelevant thoughts will lead to performance suppression (for example, Oaksford et al., 1996).

Although the model is able to predict the influence of both positive and negative moods on cognitive performance, this thesis will concentrate upon the effect of depressive or sad moods. Through theoretical and behavioural explorations, it has been hypothesised that the location for the affect-cognition interaction is the central executive component of working memory (Oaksford et al., 1996), the neurological correlates of which lie within the dorsolateral region of the prefrontal cortex (see for example, Petrides, 2000a). As well as being fundamental to the control of cognitive processing, this region of the brain has also been shown to play an important role in the experience of depressive episodes (Dolan et al., 1993) and induced depressed mood states (Drevets, 2000). This dual functionality of the dorsolateral prefrontal cortex implies a strong likelihood of being able to find neurophysiological evidence to support the Resource Allocation Model (Ellis et al., 1988).

The Resource Allocation Model makes an important distinction between the two possible influences of depressive states upon resource allocation (Ellis, 1991). The depressive mood may 'pre-empt' a portion of the total cognitive capacity, leaving sufficient resources to adequately process the criterion task (Ellis, 1991). Alternatively, there may be a quantitative reduction in the amount of resources that can be allocated to the criterion task (Ellis, 1991).

It is possible that this distinction is associated with the cognitive characteristics of the criterion task. If the task is cognitively self-contained and therefore reliant upon a single or a small number of sub-processes then sufficient resources could be allocated after the depressed mood has pre-empted its division of resources. However, should the task be cognitively intensive and require a number of sub-process for successful completion, then the quantitative reduction in resources caused by the depressed mood would lead to an insufficient allocation to the criterion task.

Furthermore, the Resource Allocation Model predicts that mood congruent memories elicited by depressed moods will only interfere with a criterion task when two conditions are met (Varner et al., 1998). Firstly that the material within the criterion task must contain at least two distinct categories of information and secondly, the thoughts produced must be relevant to one category of information and irrelevant to the other (Varner et al., 1998). Again, it is possible that these criteria can be applied to the cognitive intensity of the criterion task. Therefore a depressed mood will cause interference when the criterion task must be processed by two or more sub-processes and that the thoughts are relevant to one of the sub-processes and irrelevant to the other.

This interpretation is alluded to within the literature during the development of the Resource Allocation Model:

“Negative or disruptive states can interfere with the encoding of information as it pre-empts some capacity or resources which would normally be allocated to the process of encoding the criterion task”  
(Ellis, 1985, p393).

The aim of this thesis is investigate the effects of manipulating cognitive complexity in tasks associated with frontal activity, and further investigate mood modulation of these effects at both the behavioural and cognitive level. Findings will principally be interpreted in terms of the Resource Allocation Model.

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## Chapter 2

### Affective Neuroscience – The role of electrophysiological techniques

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#### 2.1 Neuroimaging Research<sup>1</sup>

Over the last ten years there has been a significant shift in the methodologies implemented in the study of affective neuroscience. In particular there has been an increase in the use of haemodynamic neuroimaging techniques, such as positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) (Tagamets & Horwitz, 2000). The surge in use of these techniques is associated with the superior spatial resolution (~1mm) they display (Korvenoja, Aronen, & Ilmoniemi, 2001). For example, high strength MRI scanners have been associated with resolution levels that are capable of locating activation at the cortical column level (Menon, Ford, Lim, Glover, & Pfefferbaum, 1997; Kim, Duong, & Kim, 2000).

The spatial specificity afforded by haemodynamic techniques have provided a means of localising the neuronal generators associated with human cognition (Horwitz, Tagamets & McIntosh, 1999). Unfortunately the interpretation of this data into a meaningful model of the temporal dynamics of the neuroanatomical correlates of cognitive processes is not

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<sup>1</sup> This thesis describes the empirical investigation of the influence of mood on cognition, as outlined in Chapter 1, using electrophysiological techniques. By way of professional development and to develop a sound theoretical knowledge of electrophysiological techniques, a BTEC in Clinical Neurophysiology was undertaken (Technical Appendix 1).



straightforward. Firstly haemodynamic techniques are indirect measures (Tagamets et al., 2000), which indicate changes in regional cerebral blood flow (rCBF) in PET and blood oxygenation levels in fMRI. Therefore they index metabolic changes related to brain activation, rather than directly measuring synaptic activity within neuronal populations.

Secondly, the experimental designs utilised in both techniques favour the measurement of relative changes between brain states (Tagamets et al., 2000). The majority of studies are reliant upon what are referred to as subtraction paradigms (Posner, Peterson, Fox, & Raichle, 1988), and therefore report changes in brain activation during a hypothesis-sensitive task when compared to a control task. Consequently it is difficult to apply these results to the neuronal activity which underlies the temporal dynamics of information processing.

Finally, the poor temporal resolution of haemodynamic techniques ( $> 1$  sec) (Dale & Halgren, 2001; Buxton, Wong, & Frank, 1998) means that the sequential activity within a single neuroanatomical area maybe disregarded (Halgren, Boujon, Clarke, Wang, & Chauvel, 2002). This inadequacy can be re-addressed is by utilising the superior temporal attributes of electrophysiological techniques (Deldin, Deveney, Kim, Brooks, & Best J L, 2001).

## **2.2 Electrophysiological Techniques**

The basis of the all electrophysiological techniques is the electroencephalogram (EEG). Through electrodes placed upon the scalp, the electrical activity of the brain is recorded to produce a waveform which represents changes in voltage over time. This allows this signal to be quantified in terms of cycles per second, measured in Hertz (Hz) and amplitude,

measured in microvolts ( $\mu\text{V}$ ). From this basic recording the spontaneous activity of the brain can be analysed using quantitative electroencephalographic techniques, or the signal can be time locked to a specific event in order to produce an event-related potential.

### **2.2.1 Quantitative Electroencephalography**

During this technique, the EEG signal is mathematically analysed in terms of frequency and amplitude, to provide a qualitative assessment of spontaneous brain activity. Traditionally, the frequencies of activity are classified into the bandwidths: delta (0.1-4 Hz), theta (4-8 Hz), alpha (8-12Hz), beta (12-30 Hz) and gamma ( $\sim$ 40 Hz) (Krause et al., 2000). Importantly, the simultaneously recorded activity within these bands differs (Krause et al., 2000), thereby providing a means of inferring the parallel existence of multiple mental states and/or processes (Boiten, Sergent, & Geuze, 1992; Dumont, Macchi, Carrier, Lafrance, & Hébert, 1999; Klimesch, Schimke, & Schwaiger, 1994; Klimesch, Russegger, Doppelmayr, & Pachinger, 1998).

Assessment of the different bands of activity allows the deconstruction of the scalp recorded activity in a number of ways. For example, the different frequencies have been localised to distinct neuronal generators (Fernández et al., 1998). For example, frequencies within the theta band are associated with hippocampal activation (Burgess & Gruzelier, 1997) and alpha frequencies are thought to be generated by corticothalamic and thalamocortical pathways (Klimesch, 1997; Steriade, Gloor, Llinas, Lopes da Silva, & Mesulam, 1990). Alternatively functional relationships between frequency bands with cognitive processes and arousal states can be investigated (Krause et al., 2000). Episodic memory has been associated with theta oscillations (Klimesch, 1996), while the relative power of theta and alpha activity provides an index of task demands (Klimesch, 1999).

### **2.2.2 Event-related Potentials**

If the EEG signal is time locked to a stimulus then a more detailed picture of brain processes can be produced (Rugg & Coles, 1996). During the production of event-related potentials (ERPs), the EEG signal elicited by a specific stimulus is recorded over number of trials. When these trials are averaged together, activity that is specific to the stimulus will be summated while the surrounding noise will be filtered out.

The fine temporal resolution of event-related potentials means that the distinct stages of signal processing can be differentiated. The waveform components occurring within the first 100ms post stimulus presentation are thought to be primarily sensory, reflecting the activity of the brainstem, thalamus and primary sensory cortex (Allison, 1986). Consequently, these components are sensitive to the physical characteristics of the stimulus (Stockard, Stockard, & Sharborough, 1986; Chiappa, 1983). Those components which occur in the waveform after the 100ms post-stimulus period are thought to represent the cognitive aspects information processing, for example attention and decision making (Pritchard, 1981; Picton, Donchin, Ford, Kahneman, & Norman, 1984).

In the same way that reaction time data can be deconstructed in order to provide information on response formation and execution, analysis of the waveform components within the latter sections of the ERP provides a means of representing the temporal, activational, topographic and lateralisation characteristics of information processing. Each component will have a distinct latency, amplitude, morphology and topographic distribution across the scalp that can contribute towards creating a neurological signature of cognitive processes.

### 2.2.2.1 ERP components reflecting cognitive processes

The principal use of event-related potentials is to index cognitive performance, consequently this area has been the subject of some major reviews (see for example (Rugg et al., 1996). There are a number of problems associated with attaching labels to components within event-related potentials, for example the possible confusion caused by using the latency within a notation when it actually reflects a component whose latency can occur over a wide time range (Regan, 1989). However, idiosyncratic nomenclature continues to be used in spite of calls for a standardised system, such as Picton et al (2000) ((Regan, 1989). Some of the main components associated with cognitive processes are outlined below.

#### *2.2.2.1.1 Contingent negative variation (CNV)*

Originally referred to as an expectancy wave (Walter, Cooper, Aldridge, McCallum, & Winter, 1964), this component reflects a slow negative wave which develops during the anticipatory interval between the presentation of two temporally related stimuli (Nagai et al., 2004). The component is generated over several seconds following a warning stimulus, when a second imperative stimulus is expected to initiate a reaction time response (Nagai et al., 2004).

The CNV component is traditionally divided into two separate waveforms (Hillman, Apparies & Hatfield, 2000). The initiation of the negativity at midline frontal electrodes, referred to as the O-wave, is thought to represent an orientation response (Gaillard & Perdok, 1979; Loveless, 1979). The later E-wave was originally associated with motor readiness, however more recent explanations describe the presence of stimulus preceding negativity (SPN), which is thought to reflect anticipatory attention and information

processing (Brunia, 1988; Brunia & Haagh, 1986; Ruchkin, Sutton, Mahaffey & Glasser, 1986).

#### *2.2.2.1.2 P300*

The P300 is associated with basic information processing mechanisms (Polich & Kok, 1995). Rather than being sensitive to the physical characteristics of the eliciting stimulus, the P300 is thought to be influenced by the information contained within the stimulus and the context of presentation (Squires, Duncan-Johnson, Squires, & Donchin, 1975a; Sutton, Braren, Zubin, & John, 1965). The presentation of a rare target stimulus within a sequence of standard stimuli elicits a positive deflection, maximal over parietal regions, at around 300ms post stimulus presentation (Polich et al., 1995).

It has been discovered that there are a number of components which share the characteristics described above: the classic P300 (Sutton et al., 1965), the P3a and P3b (Squires, Squires, & Hillyard, 1975b) and the Novelty P3 (Courchesne, Hillyard, & Galambos, 1975). Controversy continues as to whether these are members of one family or if they constitute distinct components (Spencer, Dien, & Donchin, 1999). However, the whole P300 family are associated with stimulus identification and the reconstruction of context-specific information (Regan, 1989). For example, the amplitude of the P300 component is thought to be sensitive to a triad of factors, referred to as the Triarchic Model (Johnson, 1986; Johnson Jr, 1993). The three influencing factors are information transfer, subjective meaning and subjective probability, the later being modulated by the transference of information (Salisbury, Shenton, & McCarley, 1999).

#### *2.2.2.1.3 N400*

The N400 component is an index of incongruity, and although originally associated with semantic stimuli only (Kutas & Hillyard, 1980), it is elicited by other examples of relationship violations (Kutas & Kluender, 1994). In the original study participants were presented with sentences, one word at a time, where the last word was either predictable (“It was his first day at work”) or unpredictable (“He spread the warm bread with socks”). The presentation of the incongruous word elicited a negative deflection at around 400ms post-stimulus, which was maximal over central-posterior regions (Kutas et al., 1980).

#### *2.2.2.1.4 P600*

The P600 component has been argued by some researchers to be the most robust reflection of syntactic processing (Osterhout, McKinnon, Bersick, & Corey, 1996), and is therefore sometimes referred to as the “Syntactic Positive Shift” (Hagoort, Brown, & Groothusen, 1993). Alternatively, it is thought to be indicative of more general cognitive processes (Coulson, King, & Kutas, 1998b; Coulson, King, & Kutas, 1998a; Gunter, Stowe, & Mulder, 1997). However, regardless of its specificity towards syntactic operations, the P600 component is thought to be associated with revision processes and working memory capacity (Friederici, Steinhauer, Mecklinger, & Meyer, 1998; Gunter et al., 1997; Münte, Szentkuti, Wieringa, Matzke, & Johannes, 1997; Osterhout et al., 1996; Osterhout, Holcomb, & Swinney, 1994; Mecklinger, Schriefers, Steinhauer, & Friederici, 1995).

#### 2.2.2.2 Interpretation of ERPs.

The millisecond temporal resolution possible with ERP data offers the possibility of tracking the time course of different components of complex cognitive processes. Changes in the amplitude of specific components can be interpreted as indices of the allocation of

specific cortical resources to the task, with increases associated with stronger electrical fields, and decreased activation or reduction in amplitude associated with a deficit in the allocation of resources (Kayser et al., 2001). It is also possible that increasing amplitude is associated with wider recruitment of underlying cortical areas (Kayser et al., 2001).

The spatial resolution possible with ERP measures is poor and the inverse problem prevents accurate spatial localisation of the source of the signals measured at the scalp. Various source localisation techniques have been devised to estimate the location of underlying neuronal generators, such as BESA (Scherg & Picton, 1991) and LORETA (Pascual-Marqui, Michel, & Lehmann, 1994), but the assumptions underlying such techniques remain challenged (Picton et al., 2000b). In general, comparisons can be made between the structural and topographical characteristics of specific components in different conditions to investigate the effects of the independent variables of interest.

### **2.3 The Use of Electrophysiological Techniques in Affective Neuroscience**

As stated in Chapter 1, the affect-cognition interaction has been located to the dorsolateral prefrontal cortex (Oaksford et al., 1996). The Resource Allocation Model (Ellis et al., 1988) predicts that the extent of the interaction is controlled by the allocation of cognitive resources under different mood states. This specificity to cortical activation lends itself to electrophysiological research as, rather than inferring cognitive activation through secondary measures such as reaction time, the degree of cortical activation can be directly assessed.

### **2.3.1 EEG Measures of Cortical Activation in Depression**

A number of different indices of cortical activation have postulated that individuals suffering from depression exhibit cerebral hypoactivity (Sara et al., 1994):

- Blunted responses to pharmacological challenges, such as cortisol (Baldessarini, 1986; Carrol, 1982);
- A reduction in electrodermal activity recorded (Ward, Doerr, & Storrie, 1983);
- A reduction in pupil responsiveness (Janisse, 1976).

Additional evidence comes from neuroimaging techniques such as Single Positron Emission Computerised Tomography (SPECT), rCBF, fMRI and PET which have demonstrated cerebral blood flow and metabolism reductions in depressed individuals (Baxter et al., 1983; Baxter et al., 1985; Brodie et al., 1983; Gur et al., 1984; Gustafson et al., 1981; Mathew et al., 1980; Phelps et al., 1985; Silfverskiold et al., 1979; Uytendhoef et al., 1983). However, the strongest evidence comes from electrophysiological measures of cortical activity.

The theoretical basis of this area of research is the postulation that there are two separate neuronal circuits involved in two major forms of motivation and emotion, approach and withdrawal (Davidson, 1998a; Gray, 1994; Lang, Bradley, & Cuthbert, 1990). The approach system is associated with appetitive behaviour and is implicated in the generation of positive affect, while the withdrawal system controls retreat from aversive stimuli and is related to the experience of negative affect (Davidson, 1998b). The neural substrate of depression is thought to be reflected in asymmetries within the prefrontal cortex that lead to disruptions to these two emotional systems (Davidson, 2003). The presence of reduced



activation within the left prefrontal regions is associated with deficits within the approach system and reduction within the right prefrontal regions is related to malfunction of the withdrawal system (Davidson, 1994; Davidson, 1998a).

#### 2.3.1.1 Anterior Asymmetry and Emotion

Patterns of electrical activity within the frontal lobes have been related to a broad range of individual differences in emotional reactivity, referred to as affective style (Davidson, 1998a). For example, 10 month old babies who cried upon maternal separation were shown to have less left and more right prefrontal activation during the period preceding the separation test, when compared with babies who did not cry in these circumstances (Davidson & Fox, 1989). Furthermore, adults who showed baseline EEG asymmetries in prefrontal activation showed differential profiles of positive and negative affect, as measured by the trait version of the Positive and Negative Affect Scales (PANAS; Watson, Clark, & Tellegen, 1988). Those who exhibited left-frontal activation were associated with a mood profile of high levels of positive affect and low levels of negative affect, while the mood profile of those exhibiting the opposite asymmetry reflected low levels of positive affect and high levels of negative affect (Tomarken, Davidson, Wheeler, & Doss, 1992).

This pattern of EEG asymmetry is also associated with the psychopathological experience of emotions, with left hypofrontality being recorded in patients suffering from depression (Henriques & Davidson, 1990; Henriques & Davidson, 1991; Schaffer, Davidson, & Saron, 1983). This relationship has been extensively investigated, and a number of researchers failing to replicate the reduction of left prefrontal activity in depression (see for example Reid, Duke, & Allen, 1998). However, Davidson (1998) reported that many discrepancies are a result of differences in EEG methodology, and comments that studies such as that by

Reid et al. (1998) have more in common with the model of anterior asymmetry and emotion than the authors suggest.

### **2.3.2 Event-related Potential Measures of Cortical Activation in Depression**

Much of the research utilising event-related potentials to investigate the electrophysiological correlates of depression have focused upon the P300 component. A reduction in P300 amplitude has been reported in depression (Diner, Holcomb, & Dykman, 1985; Gangadhar, Ancy, Janakiramaiah, & Umapathy, 1993; Muir, St Clair, & Blackwood, 1991; Shagass, Roemer, Strumanis, & et al, 1981), while other studies have shown no difference in amplitude between depressed patients and healthy controls (Bruder et al., 1991; El Massioui & Lesèvre, 1988; Giedke, Bolz, & Heinmann, 1980; Have, Kolbeinsdon, & Péursson, 1991; Plooijs-Van Gorsel, 1984; Kaustio, Partanen, Valkonen-Korhonen, Viinamäki, & Lehtonen, 2002; Sara et al., 1994). Although there is no consistent pattern of event-related activity in depression, a clearer picture has been found in other psychopathologies. For example, the reduction in amplitude associated with patients suffering from schizophrenia is thought to reflect malfunction at the level of the neuronal generators of the P300 (Salisbury et al., 1999).

The application of event-related potentials allows the investigation of the temporal and, to some degree, the spatial characteristics of the cortical correlates of information processing. Changes in waveform activity can be interpreted in terms of the less successful allocation of resources (for example Kayser et al., 2001) and are therefore a valuable tool in the investigation of the nature of the affect-cognitive interaction which is the basis of this thesis.

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## Chapter 3

### Methodological Considerations

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#### 3.1 Mood Induction Techniques

##### 3.1.1 Introduction

Moods and emotions have always been a fascination to psychologists, whether the aim is to understand the theoretical basis of emotions or to investigate how moods influence behaviour. However, no matter how extensive the scope of the research there is one connecting factor, the experimenter must be able to treat the mood as an independent variable. The use of clinical populations in mood research is associated with the risk of co-morbidity, for example depression is often co-morbid with anxiety (Lane, Carroll, Ring, Beevers, & Lip, 2003). Furthermore, the wide range of symptoms associated with major depressive disorder (Akiskal, 2000) could result in a situation where the profiles of patients within the clinical group differ significantly.

To overcome these problems induced moods are often used in mood cognition research. Two main approaches have been established, one which utilises naturally occurring mood states and the second which actively manipulates the participant's mood.

##### 3.1.1.1 Utilising Naturally Occurring Moods

There are three ways in which natural mood states have been used in mood research. The first involves screening participants at the time of the experiment and placing them in

appropriate conditions (Hettner & Ballif, 1981). The second is based on the assumption that certain circumstances are consistently associated with particular mood states, for example conducting the experiment on rainy and sunny days (Parrott & Sabini, 1990; Schwarz & Clore, 1983), or after examination results (Parrott & Sabini, 1990; Russell & McAuley, 1986). Finally, as mentioned previously, comparisons can be made between clinical and non-clinical populations (Weingartner et al., 1981). Unfortunately, all three methods are associated with a number of problems, the main one being confounding variables.

If the experimental manipulation is relying on the participant's natural mood state at the time of the experiment, the main confound is whether the mood is state or trait (Martin, 1990). Therefore there will be no indication whether any experimental effects are the result of the individuals underlying personality traits or a consequence of the current mood state (Martin, 1990). The list of confounding variables increases when clinical populations are used. Possible confounds include pharmacological intervention, varying therapeutic regimes and the effects of institutionalism (Martin, 1990).

Another important factor is the degree of control the experimenter has over the timing of the mood. Take for example, a study investigating whether mood had a differential effect on encoding and retrieval in memory. If the participant's natural mood is used, regardless of whether they fall into a clinical or non-clinical population, their mood will be present throughout the experiment (Martin, 1990). As a result a comparative investigation would not be possible.

#### 3.1.1.2 Mood Manipulation

Consequently, attention has fallen onto the active manipulation of participant's mood (Philippot, 1993). By adopting an empirical approach, the control and precision which is required during the investigation of complex emotions such as depression and happiness can be achieved (Brewer et al., 1980).

### **3.1.2 Mood Induction Procedures**

To evaluate the different mood induction procedures currently used in mood research, the methodological procedures of 173 studies have been analysed (Appendix 1). The remainder of this section will outline the procedures and describe the criteria that must be fulfilled if the procedures are to be successful.

#### 3.1.2.1 Statements

It is generally accepted that statement based procedures are the most commonly used method of mood induction (Goodwin & Williams, 1982). In this review, 38% of the studies examined used this technique. The basis of this procedure involves experiencing the mood suggested by statements designed to elicit elated, depressed and neutral moods. The most common example is the Velten Mood Induction Procedure (VMIP; Velten, 1967; Velten, 1968).

#### 3.1.2.2 Imagination

Of the 173 studies, 15% fell into the imagination based procedure category. The most common example was the autobiographical recall technique (Brewer et al., 1980), where participants recall autobiographical events with the aim of inducing either happy or sad moods. The other version involves the participant being instructed to empathise with an

emotive story and imagine how they would feel in similar circumstance, for example having a friend diagnosed with an incurable disease (Williams, 1980).

### 3.1.2.3 Film

Films have been known to produce an emotional response since their employment in the study of stress (Goodenough et al., 1975). Although film was used to induced moods in 13% of studies, there was little consistency in the type of film used and many relied on study specific film clips (for example: Oaksford et al., 1996; Schotte et al., 1990; Isen et al., 1987; Isen & Gorgoglione, 1983). To address this problem, Gross & Levenson produced a set of films which could elicit the discrete emotions amusement, anger, contentment, disgust, fear, neutral, sadness and surprise (Gross & Levenson, 1995). A similar set of films was produced by Philippot (1993), however the Gross & Levenson (1995) collection is more widely used.

### 3.1.2.4 Music

Used in 9% of studies, this procedure uses suggestive music as a framework to achieving a depressed, elated or neutral mood (Martin, 1990). The main variation between studies occurs in the way in which the music is presented to the participants. There are differences in the degree of choice the participant has over the music, either choosing from an appropriate list (Sutherland et al., 1982) or the same selections being presented to all (Clark & Teasdale, 1985). Variations also occur in the way in which stimulus tapes are compiled. The elation, depression and neutral tapes can either be consistent in tone (Clark & Teasdale, 1985) or they may begin with a neutral selection which progressively becomes more elating, more depressing or remains neutral with each successive selection (Pignatiello et al., 1986).

#### 3.1.2.5 False feedback

During this technique, used in 9% of studies, participants perform a task and are given feedback on their performance (Martin, 1990). To induce a positive mood they are told they performed well and for a negative mood they are told they performed badly (Forgas et al., 1990; Isen et al., 1978).

#### 3.1.2.6 Affective Pictures

Out of the 5% of studies that use affective pictures to induce mood, all but 2 used the International Affective Picture System (IAPS; (Lang et al., 1988). The IAPS is a series of colour photographs which vary along the dimensions of valence (pleasant-unpleasant) and arousal (excited-calm) (Lang et al., 1993). In the majority of experiments, these pictures are classified into positive, negative and neutral categories, within which the negative and positive pictures have equivalent arousal, while both these categories are more arousing than the neutral material (Vrana et al., 1988).

#### 3.1.2.7 Facial manipulation

During this technique, used in 4% of studies, the participant is given muscle by muscle instructions for one of six emotional facial configurations, without giving them an indication of the associated emotion (Ekman et al., 1983). For example, the instructions for the anger configuration would be: a] pull your eyebrows down and together, b] raise your upper eyelids and c] push your lower lip up and press your lips together.

#### 3.1.2.8 Mixed Procedures

It is becoming more popular to combine established mood induction procedures to produce a more robust effect. Examples include combining the VMIP with music (Dykman, 1997;

Fox et al., 1998; Slyker & McNally, 1991), or autobiographical recall and music (Gilboa-Schechtman et al., 2000; Kulbartz-Klatt et al., 1999).

### **3.1.3 Evaluation of techniques**

There are a number of criteria which should be kept in mind when assessing mood induction procedures:

- The ethical boundaries of the procedure;
- The reproducibility of the induced emotion;
- The specificity of the induced emotion;
- The duration of the induced emotion;
- The intensity of the induced emotion.

#### **3.1.3.1 The Ethical Boundaries of the Procedure**

There are two main ethical considerations that surround the manipulation of mood. First, regardless of the induction procedure employed, it must be kept at the forefront of the experimenter's mind that if a negative manipulation is being performed there is a risk of eliciting traumatic memories (Philippot, 1993). As a result it is imperative that participants are screened prior to taking part in the experiment, for example using the Beck Depression Inventory (BDI; (Beck, 1987)). It is widely accepted that if an individual score more than 9 on the BDI, then they are mildly to moderately depressed (Beck, 1987; Killgore, 1999; Spreen & Strauss, 1991) and as a result they should not take part in a mood induction study. Also to minimise the risk of eliciting traumatic memories in non-depressed participants it must be accepted that only mild emotions can be elicited in the laboratory (Philippot, 1993).



The second consideration relates to how participants are treated after the manipulation and experimental procedure has been completed. Whereas the necessity for debriefing within experimental situations based around obedience and false information is widely accepted, their use in mood manipulation procedures is far from prevalent (Frost & Green, 1982). Out of the 173 studies reviewed here, only 19% used a specific debriefing strategy. As stated in Holmes' review of debrief usage, proper debriefing can eliminate misinformation generated in the experiment and eliminate the negative after effects of such procedures (Holmes, 1976a; Holmes, 1976b). As the basis of mood manipulation is either the participant being presented with false information or focusing on emotive material, the use of an effective debrief procedure is essential.

However, a full debrief is only part of the required procedure, it is also important that the induced mood is removed. Of the studies reviewed, only 20% actively returned the participant's mood to their baseline level, the majority of whom used the elation statements from the VMIP. Frost & Green (1982) showed that instructing participants to read 30 of the statements from the elation series removed the negative effects of the depressive VMIP that would have otherwise remained.

### 3.1.3.2 The Reproducibility of the Induced Emotion

No matter how effectively a technique can manipulate moods in an individual, the important test is whether the effect is reproducible across a large number of participants. It is always a possibility that a procedure will differentially effect quantitative and qualitative aspects of mood (Philippot, 1993). For example, early techniques based on hypnosis produced strong quantitative mood changes, however only 15% of participants were susceptible (Brewer et al., 1980).

Although the claims of success for many of the techniques vary from study to study, they are all fairly successful. Success rates for the VMIP show the most variance, ranging from 50% (Polivy & Doyle, 1980) to 70 % (Teasdale & Russell, 1983). Although claims of 100% success have been made for musical MIP (Clark, 1983), most studies report a success rate of 75% (Martin, 1990). Of the remaining techniques, film and autobiographical recollection have a success rate of about 75%, while facial expression and task feedback are about 50% successful (Martin, 1990).

Following Gouaux & Gouaux (1971) suggestion that women respond more strongly to mood manipulation than men, there has been tendency to use only female participants. Although gender differences have remained a contentious issue within mood research (Westermann et al., 1996), few studies have provided support for this recommendation (Clark, 1983). Another long-standing issue is whether personality traits may bias mood induction techniques, but again this concern is unsubstantiated (Lewis & Harder, 1988).

#### 3.1.3.3 The Specificity of the Induced Emotion

In any study that relies on an induced mood, the specificity of the mood is of utmost importance. That is not to say that experiments dealing with emotions are more at risk from confounding variables, but it is not uncommon for more than the target emotion to be manipulated by a mood induction procedure (Atkinson & Polivy, 1976; Strickland et al., 1975). When it is considered that the aim is to produce an analogue of a mood which can naturally show co-morbidity, this is hardly surprising.

A natural concern is the tendency for depression to covary with anxiety and hostility (Polivy, 1981). This is highlighted by the music MIP which produces elevations in both

depression and anxiety, to the degree that anxiety can exceed despondency and sadness (Clark, 1983; Sutherland et al., 1982). The VMIP on the other hand produces a mood which is consistent with the nature of naturally occurring depressed mood states (Clark, 1983). In studies where the VMIP has been shown to have an effect on other negative moods, it has been relatively weak (Clark, 1983).

However, as long as researchers implicitly state what emotions they are interested in and make appropriate manipulation checks, this issue will not become a real problem. Unfortunately, of the 173 studies reviewed, only 58% carried out post manipulation checks. As a result, there is no indication of what mood, if any has been manipulated. It is therefore important in an experiment where conclusions as to the effect of mood on a particular attribute are made, that pre and post mood measures must be compared.

Following from this, it is important that a validated method of mood assessment is used. 36% of the studies reviewed here used study specific scales, not only does this allow room for experimenter bias, but it also makes the comparison of results difficult. A wide variety of scales exist, however the most common include the Multiple Affect Adjective Checklist (MAACL; Zuckerman & Lubin, 1965), the Positive Affect and Negative Affect Scale (PANAS; Watson et al., 1988), the Depression Adjective Checklist (DACL; Lubin, 1965), and the Visual Analogue Scale (VAS; McCormack et al., 1988).

#### 3.1.3.4 The Duration of the Induced Emotion

One factor that has been overlooked by the majority of the studies is the duration of the induced mood. None of the studies reviewed carried out a manipulation check at the end of their experimental procedure to ensure that the induced mood had lasted as long as they

were recording their dependent variable. If a long or repeated measures task was used, the conclusions drawn must be viewed with caution. Under these circumstances, there can be no clear distinction between knowing whether the mood genuinely had no effect or that it had simply dissipated (Clark, 1983).

When the few studies that have looked at duration effects are considered, the necessity for the inclusion of a final manipulation check becomes clear. When the Velten MIP and film manipulation were compared after a four minute filler task, the results were cause for concern (Isen & Gorgoglione, 1983). The positive film produced the most stable result, both after induction and the four minute delay. The positive and negative Velten procedures came next with stable results directly after induction but a reduced effect after four minutes. While the negative film, which focused on inducing an anxious mood produced no effect at (Isen & Gorgoglione, 1983). In a more specific study, Frost & Green (1982) found discrepancies between the duration of elation VMIP and depression VMIP, negative effects were present after 10 minutes whereas elation effects have disappeared (Frost & Green, 1982).


#### 3.1.3.5 The Intensity of the Induced Emotion

To appreciate the varying intensities of the induced emotion, there must be some discussion of theories of emotions that have been postulated. Over the last 15 years there has been resurgence in studying the theoretical perspectives of emotions, with the emphasis on neuropsychological and cognitive based theories (Winter & Kuiper, 1997). The neuropsychological theories aim to identify the brain systems that mediate emotions, whether at an anatomical level (Davidson & Irwin, 1999) or the motivational pathways which underlie emotions (Gray, 1990). Cognitive based theories have focused on cognitive

orientation, the idea that an individual's evaluation and interpretation of a situation will shape their emotional experience (Izard, 1993; Lazarus, 1993).

However, these theories should not be placed in opposition to one another; a complete theory of emotion is one that takes into account the neurological, psychological, visceral and cognitive elements of an emotional experience. Therefore emotions should be seen as experiences which have cognitive, somatic and psychological attributes. Take for example depression, the cognitive attribute could be the negative cognitions which are thought to have a causal role in the production of symptoms (Beck, 1967; Beck & Rush, 1978; Beck et al., 1979). The somatic attribute could be psychosomatic illness associated with the depression, for example an inability to sleep (Costin & Draguns, 1989), and finally the psychological attribute could be the individual's awareness of their sadness. It follows then that the intensity with which an emotion is experienced will depend on the relative contribution of each of these attributes.

Emotions can be seen to lie on a continuum where the intensity of experience ranges from reflective to reactive. Should an emotion have strong cognitive, somatic and emotional attributes then it will be experienced on a reflective level. However, if there are only emotional and possibly somatic attributes, then the experience will be reactive. If this process of classification is applied to the moods induced by the procedures described above, it can be seen that the resultant emotions lie on this continuum of intensity (Table 3.1).

MIP	Cognitive Component	Somatic Component	Psychological Component	Intensity of emotion
VMIP	Y	Y	Y	<div> <div>Reflective</div> <div>  </div> <div>Reactive</div> </div>
ABR	Y	Y	Y	
False feedback	Y	Y	Y	
Film	Y?	Y?	Y	
Music	Y?	Y?	Y	
Facial expression	N	Y?	Y	
Affective imagery	N	Y?	Y	

**Table 3.1** The attributes and intensity of emotions produced by the mood induction procedures reviewed (Key: Y – component present; N – component not present). Where the presence of a component is questioned, it indicates that individual differences that would influence the manipulation procedure.

Therefore, the facial expression and affective imagery techniques produce emotions with a reactive intensity. Take for example the IAPS induction procedure, if the participant is presented with a picture of a mutilated body they will have an emotional response of disgust, and possibly an associated physiological response. However, because of the transitory nature of the stimuli it is unlikely that the participant will apply any cognitive connotations to the experience and once the image has been removed they will no longer have an emotional attachment to the image. A a result, an emotion which has a reactive intensity would be induced.

The VMIP, autobiographical recall and false feedback techniques on the other hand will induce emotions that have a reflective intensity. In these techniques the participants are encouraged to dwell on the experimental stimuli and therefore have the opportunity to

internalise their reaction. As a result of this they are likely to form a more robust emotion which has cognitive attributes.

The final two procedures, film and music, would produce emotions which fall in the centre of this continuum. Any emotion induced by film and music is more likely to be modulated by the personal experiences of the participant's than any of the other techniques. If an individual can make a cognitive associations with a film clip or a piece of music it would expected that they would experience a reflective emotion. However, if the stimulus did not strike a chord with the participant they would then only experience a reactive emotion.

#### **3.1.4 Conclusion**

Regardless of the actual technique used, an induction procedure should be used with the following criteria in mind:

1. The induction of mood is an ethically sensitive technique, therefore a full debrief procedure should be used, including reversal of the induced mood, confirmation of the return to baseline mood level and an explanation of the nature of the experiment.
2. It is not uncommon for affects other than the target emotion to be manipulated by an induction procedure (Atkinson & Polivy, 1976; Strickland et al., 1975). To minimise the possibility that conclusions could be attributed to the wrong emotion, the emotion of interest should be explicitly stated.
3. Following from this, a valid manipulation check must be used to measure the success of the mood induction procedure. Also, where possible participant's pre-induction scores should be taken into account during this assessment (Clark, 1983).

4. The duration of an induced mood is generally short lived. As stated by Clark (1983), experimental procedures involving long or series of tasks should use a method of maintaining the induced mood.

When making a decision on what induction procedure should be used, the most important consideration is what intensity of emotion is required. As this thesis is investigating the effect of emotion on cognitive processes it is important that the intensity of the induced emotions is cogent. Therefore the chosen procedure must be capable of inducing reactive emotions. As the effects of individual differences should be minimised, the three possible techniques are the VMIP, the autobiographical recall procedure and the false feedback procedure. As no comparison of the three techniques has been conducted, Chapter 4 will describe a study to choose the most appropriate technique.

### **3.1 The Measurement of Mood**

#### **3.2.1 Subjective measures**

As previously stated, there are a wide variety of mood assessment scales, however the most common include the Multiple Affect Adjective Checklist (MAACL; Zuckerman & Lubin, 1965), the Positive Affect and Negative Affect Scale (PANAS; Watson et al., 1988), the Depression Adjective Checklist (DACL; Lubin, 1965), and the Visual Analogue Scale (VAS; McCormack et al., 1988).

However, due to it's connection with Davidson' (1998) anterior asymmetry and emotion model, the PANAS will be used in this thesis. This scale measures both a Positive Affect (PA) score and Negative Affect (NA) score and is widely used in mood induction studies and considered psychometrically superior to other mood measures (Wiseman & Levin,



1995). PA reflects enthusiasm and alertness, therefore a high PA score represents a state of high energy and pleasurable engagement while a low PA score is associated with sadness and lethargy (Watson et al., 1988). NA encompasses a wider feeling of distress and unpleasurable engagement, therefore a high score reflects a state of torment, contempt and nervousness while a low score is representative of serenity and calmness (Watson et al., 1988).

### **3.2.2 Objective Measures**

Although changes in heart rate are an accepted psychophysiological sequelae of emotion (Foster & Webster, 2001), the nature of the relationship remains unclear (Waldstein et al., 2000). A number of studies have reported differential heart rate responses to positive and negative stimuli (Sinha & Lovallo, 1992; Ekman et al., 1983; Schwartz et al., 1981), while increases across all valences have also been recorded (Foster & Webster, 2001; Waldstein et al., 2000; Cacioppo et al., 1993; Warner & Strowman, 1994).

It is possible that differential effects in response to positive and negative stimuli are related to the cortical innervation of autonomic responses (Waldstein et al., 2000). A connection has been made between evidence of the asymmetrical expression of emotion in the frontal lobes (Davidson, 1992; Hagemann et al., 1998) and the lateralisation of the control of cardiovascular reactivity (Lane & Schwartz, 1987; Waldstein et al., 2000). Alternatively, indiscriminate heart rate increments may reflect a generalised emotional response to affective stimuli or a state of task associated arousal (Cacioppo et al., 1993; Warner & Strowman, 1994; Nyklicek et al., 1997).

### **3.3 Dense Array Electrophysiological Techniques**

#### **3.3.1 Background**

Contemporary neuroimaging techniques, such as functional magnetic resonance imaging (fMRI), allow the mapping of the brain with fine structural resolution. However, as these techniques rely on metabolic and vascular processes there is a limitation as to how much temporal information can be gained (Tucker, 1993). Conversely, recordings of the electrical fields generated by the brain have excellent temporal resolution and are therefore ideal for examining the dynamics of cortical function (Tucker, 1993). Unfortunately their utility is constrained by the absence of information on the source of the recordings (Tucker, 1993).

Standard electrophysiological techniques use the 10-20 international electrode placement system (Jasper, 1958). Using the bony protrusions of the skull as landmarks, such as the nasion and inion, the electrodes are placed proportionally over the scalp along measurement lines. Although this revolutionised electrophysiological practice by standardising electrode placement, it places a limit on the number and location of measurement sites (Tucker, 1993). For electroencephalography to overcome the limitations imposed by the lack of spatial resolution, a method of recording is required where electrodes are evenly distributed across the scalp (Tucker, 1993).

#### **3.3.2 Dense Array Recordings**

In choosing the appropriate array of electrodes, the main consideration is the avoidance of aliasing. During the digital recording of the electroencephalogram (EEG), temporal aliasing is avoided by sampling at a frequency greater than the Nyquist frequency (Bendat & Piersol, 1971). To avoid spatial aliasing, inter-electrode distances must be less than 3 cm

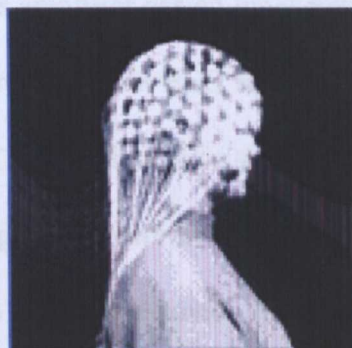
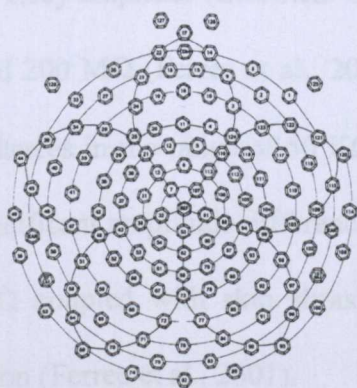
(Gevins, 1990; Spitzer et al., 1989). Therefore the array must contain many more electrodes than used in the 10-20 system (Tucker, 1993).

By using such a dense array, the optimal data set for source localisation algorithms is created (Tucker, 1993). The volume conduction properties of the brain mean each electrical source creates a dipole field which conducts throughout the head volume (Nunez, 1981). A dense array of electrodes across the scalp will ensure the entire potential field is sampled. Where electrodes provide minimal information on the potential fields, they will contribute to the noise estimations which are an essential component of source localisation algorithms (Tucker, 1993). Therefore the use of a dense array provides an easy to use and relatively inexpensive method of achieving both spatial and temporal resolution whilst recording brain activity.

The main practical issue is to find a way of applying such an array across individuals. In each case there will be a specific distance between the sensors which will allow the array to be evenly distributed across the scalp. As it would be impractical to calculate this distance and individually apply the electrodes each time a recording is made, an application device which approximates these distances is required (Tucker, 1993). One such device is the Geodesic Sensor Net (GSN; (Tucker, 1993), a 128-channel dense sensor array which will be used to record all electroencephalographic activity reported in this thesis (Figure 3.1).

The GSN employs a tension network design which allows uniform distribution of the sensors whilst holding them in position against the head (Tucker, 1993). The tension is maintained in the same way that an even surface tension exists in soap bubbles, it is

**Geodesic Sensor Net**  
128 Channel V2.0



**Figure 3.1** The layout of sensors in the geodesic sensor net and the net in position on a participant.

directed towards the centre of the sphere. This is achieved practically by applying the tension in straight lines between pairs of sensors. Therefore each straight line in the sensor net is a geodesic, that is the shortest possible distance between two points on a sphere (Tucker, 1993). As a result, the geodesics form a network of triangles which approximate the spherical surface of the skull (Tucker, 1993)

### 3.3.3 Practicalities of Using the GSN

Traditionally scalp-electrode impedance levels of less than 5 K $\Omega$  are maintained during electrophysiological recordings (Ferree et al., 2001). For these levels to be attained, skin abrasion is required to remove the surface epidermal layer which has a naturally high impedance (Ferree et al., 2001). Unfortunately, this procedure is associated with the risk of infection from blood-borne pathogens such as human immunodeficiency (HIV), hepatitis C virus (HCV) or Creutzfeldt-Jacob disease (CJD) (Ferree et al., 2001; Tucker, 1993). However, the GSN and its associated acquisition hardware has been designed so that scalp abrasion is no longer necessary (Ferree et al., 2001; Tucker, 1993).

The signals recorded with the GSN are amplified with a high input impedance Net Amps dense array amplifier (Electrical Geodesics, Inc). This amplifier has an input impedance of around 200 M $\Omega$  (Ferree et al., 2001) and is able to record clean EEG signals with sensor impedances in the range of 50 K $\Omega$  (Tucker, 1993). Comparisons have shown that there is no significant amplitude difference between recordings made with impedances of less than 10 K $\Omega$  coupled with skin abrasion and recordings with impedances of 40K $\Omega$  and no abrasion (Ferree et al., 2001).

As a result of this impedance range, the GSN utilises silver/silver chloride electrodes which are embedded in a sponge (Tucker, 1993). Prior to recording, the GSN is simply soaked in a potassium chloride (KCl) solution. Therefore, not only does the participant avoid the painful skin abrasion process, but also the messy aftermath which accompanies the use of electrode gel.

A possible disadvantage of the GSN is the generation of low impedance bridges (Tenke & Kayser, 2001). Over application of the KCl solution can occur when it is applied to individual sensors when the desired impedance levels are being achieved. Should this occur, the resultant electrolyte leakage can cause the creation of low impedance brigs between sensors (Tenke & Kayser, 2001). These bridges as a cause for concern as they can cause artificial similarities in event-related potentials (ERPs) recorded at neighbouring sensors (Tenke & Kayser, 2001). Fortunately the concentration of KCl used is unlikely to cause impedance bridges (EGI 2001). However, as it cannot be assumed that they will not occur, an algorithm has been developed which can successfully detect them (Tenke & Kayser, 2001).

### **3.3.4 Regions of interest**

Due to the amount of data generated within dense array recordings, it is common for regions to be used in order to gauge the topographic distribution of activation (Curran, Tucker, Kutas, & Posner, 1993). In the same way that averaging EEG data in the production of event-related potentials removes spontaneous activation and preserves activation associated with specific task processes, average the ERP across regions of interest 'cleans up' the data (Curran et al. 1993).

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## **Chapter 4**

### **Mood Induction Procedure Study**

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#### **4.1 Introduction**

Of the seven mood induction techniques described in Chapter 3, three have the capability to consistently induce moods of a reflective intensity. They are the Velten mood induction procedure, the autobiographical recall mood induction technique and false feedback mood induction technique. Although the techniques are procedurally different, they share a theoretical common ground. In each case, the central premise is that the induced mood will occur by altering the individual's cognitive perspective. As a result, the induced mood will have active cognitive, somatic and cognitive components and therefore will be of a reflective intensity. Although these procedures have not been systematically compared, their individual characteristics have been investigated.

##### **4.1.1 The Velten Mood Induction Procedure (V-MIP)**

This procedure was created during an investigation of how individual's interpretations mould their affective responses (Velten Jr., 1967; Velten Jr., 1968). The theoretical foundation of Velten's work was the work of Ellis (Ellis, 1963), who believed that statements based upon irrational beliefs would result in emotional upset. If this were true, it would provide support for the semantic psychotherapy favoured by Velten (Velten Jr., 1968), as an individual's behaviour could be altered if the evaluative statements were modified (Brewer et al., 1980).

To test his hypothesis, Velten developed a number of self referential mood statements, which were designed to induce elated, depressed and neutral moods. The aim was not only to provide support of his theoretical stance within psychotherapy, he also wanted to discover a technique similar to hypnosis, so that a person could "talk himself into a mood" (Velten Jr., 1967). Velten was aware of potential use of his technique as a means of inducing emotion in the laboratory (Velten Jr., 1968) and the statements are now widely viewed as the most common method of mood induction (Goodwin & Williams, 1982).

The V-MIP procedure involves reading and experiencing the mood suggested by a series of 60 self-referential statements. The statements of the elation treatment (E-VMIP) are designed to create a feeling of happiness, liveliness and optimism (Velten Jr., 1967). Statements include:

"If your attitude is good, then things are good, and my attitude is good."

"This is great - I do feel good - I am elated about things."

The tone of the depression treatment (D-VMIP) is one of tiredness, unhappiness and pessimism (Velten Jr., 1967). The 60 statements in this series can be divided into two categories, those which describe the somatic states associated with depression and those which focus on self-devaluation (Riskind et al., 1982). Examples include:

"Every now and then I feel so tired and gloomy that I'd rather just sit than do anything."

"I have too many bad things in my life."



The final series of statements, the neutral treatment (N-VMIP), were designed to induce a neutral mood and thereby act as a control in Velten's experimental work (Velten Jr., 1967).

Examples include:

"Some streets are said to be still listed under their old names."

"When the Banyan tree bent down under its own weight, its branches began to take root."

Although widely used, one criticism consistently levied against the VMIP is the possibility that participants are responding to demand characteristics. That is, as there is an awareness that the experimenter is trying to manipulate mood, any effects seen are as a result of the participant trying to fulfill the aims of the experiment rather than a true manipulation of mood. Velten himself was aware of this potential confound and consequently included the demand control treatments: elation demand characteristics (EDC) and depression demand characteristics (DDC) (Velten Jr., 1968). Participants were told of the procedure used in the EL (or DE) treatment and prior to being shown five samples of the EL (or DE) statements were told to behave the way they would estimate a person to behave after reading 60 of the EL or DE statements (Velten 1968). These two methods were unsuccessful at inducing elation or depression, leading Velten to conclude that the participants did not respond to the demand characteristics of the experiment (Velten 1968).

Not everyone believed that Velten's control group satisfactorily ruled out demand characteristics. The main concerns centred on the instructions given to the subjects. In their assessment, Polivy & Doyle (1980) replicated Velten's study but altered the instructions given to the demand characteristic control groups. Their participants were informed that

people often feel the opposite of the emotion expressed to them, therefore if they responded to demand characteristics they would express the opposite emotion to the statements (Polivy & Doyle 1980). Although no strong reversal effects were found, Polivy & Doyle felt that Velten's confidence in his procedure was misguided and there was in fact evidence of demand characteristics.

Although Polivy & Doyle (1980) were unable to conclude that participants were responding to demand characteristic, the controversy remained and the focus returned to discrepancies in Velten's (1968) demand control instructions (Buchwald et al., 1981). It was felt that as the EDC and DDC conditions did implicitly instruct the participants to behave in a particular manner, it was hardly surprising that no demand characteristics were found (Buchwald et al. 1981). To control for this, the participants in the demand control conditions were given a brief description of an elated or depressed mood prior to being shown five example statements from the Velten series. As no significant differences were found between the treatment and demand conditions, Buchwald et al. (1981) felt the V-MIP could not be recommended as a mood induction technique.

The majority of studies however, do not agree with this strong negative stance. It is generally accepted that, as the Velten instructions explicitly ask participants to respond to the statements as if they are their own thoughts, demand characteristics are an associated risk (Lewis & Harder, 1988). In light of this, the emphasis shifted to minimising the effects of demand characteristics rather than discussing their existence. In an attempt to do this, Larsen & Sinnett (1991) carried out a meta-analysis of 341 Velten studies to see if procedural details influenced the efficacy of the technique.

The main influencing factors were the cover story used and the method of mood assessment employed. The largest manipulation effects were seen in experiments where an honest cover story and self-report assessment were used (Larsen & Sinnett, 1991). Although this appeared to support the argument that participants were simply responding to demand characteristics, this did not lead them to agree with Buchwald et al. (1980). They concluded that "demand conditions probably serve to somewhat inflate an already significant mood induction effect" (Larsen & Sinnett, 1991), p331). As a result they suggested using a procedure including a deceitful cover story coupled with non-self-report mood assessment to minimise the effects of demand characteristics (Larsen & Sinnett, 1991).

The main limitation of this conclusion is that all methods of self-report were grouped together during the analysis, therefore placing validated scales along side a vast number of study specific scales. Although Larsen & Sinnett (1991) accept that these scales may differ in sensitivity, they do not acknowledge the degree of design bias that can be introduced by the presence of study specific scales. Therefore, the type of self-report assessment could have produced the effect rather than self-report per se. Consequently a validated self-report scale, used in conjunction with an objective measure of mood would be more appropriate.

Along side the changes proposed to minimise demand characteristics, a number alterations to the procedural detail of the V-MIP have been proposed. Some modifications simply reflected changes in social concerns, for example the statements have been updated to remove references to the Vietnam war (Polivy & Doyle, 1980). While other variations have focused on the use of the statements, for example the free association technique developed by Seibert and Ellis (1991). During this procedure, the participant is presented with 25 self-referential statements and is instructed to free associate with any thoughts which arise as a

result (Ellis et al., 1997). This technique was favoured by the team as it produced comparable results to the V-MIP while using contemporary language and avoiding references to suicidal and somatic states (Varner & Ellis, 1998; Ellis et al., 1997; Beck & McBee, 1995).

Further proposals have arisen from investigations into the theoretical basis of depression. As previously stated the Velten depressive statements are split into two categories, those describing somatic states of depression and those focusing on self-devaluation. As there are potential implications for therapeutic treatment, much interest has surrounded possible differential effects of these two categories.

Firstly, Sherwood, Schroeder, Abrami & Alden (1981) made a direct comparison of the mood induced by self-referential and non-self-referential statements. Their study showed only the statements which focused on the self-referent constructs of elation and depression successfully manipulated mood. As this was achieved with only 15 statements, they concluded “mood states may be manipulated in a more efficient and economical manner than has been heretofore realised” (Sherwood et al., 1981, p 107).

Although the implications for experimental design were highly beneficial, the results were never replicated. In a similar experiment, Riskind et al. (1982) used the original Velten series, a self-devaluation statement only series and a somatic state statement only series. They found that all three treatments produced moods that significantly differed from the elation treatment (Riskind et al., 1982). Also, there is some doubt as to how robust the mood induced in the Sherwood et al (1981) study actually was as mood assessment were only made directly after manipulation. When a mood assessment was made 6 minutes after

induction during a comparison of the full Velten series with a 25 statement version, only the full series produced a mood which lasted the extended duration (Schare & Lisman, 1984).

As a result, the full 60 statement series developed by Velten (1967) will be used in this study. The only modifications made will be to update any statements which reference events specific to the 1960s and remove any American slang and replace with British alternatives.

#### **4.1.2 The False Feedback Mood Induction Procedure (FF-MIP)**

The theoretical basis of the false feedback mood induction procedure (FF-MIP) is the work on the affective consequences of achievement proposed by Weiner and his colleagues (Weiner et al., 1979; Weiner et al., 1978). This theory predicts that people will experience positive or negative emotions as a consequence of success and failure respectively (Weiner et al., 1979; Weiner et al., 1978).

When applied to mood induction, participants are led to believe that they are performing better or worse than their true performance level in order to instigate the appropriate mood. For example, Forgas, Bower and Moylan (1990) used a sentence completion task (e.g., "car is to road as train is to .....") to provide participants with bogus information on their performance. In the negative treatment condition, participants were told that "people find these questions quite easy and completed all items in less than 5 minutes". Alternatively, participants in the positive treatment condition were led to believe that "people rarely completed more than 10 items in 5 minutes" (Forgas et al., 1990). After scoring, the participants undergoing the negative treatment were told they performed below average,

while those in the positive treatment were informed they had performed above average. As a control, the third group of participants were given no feedback and just thanked for their help in the development of a new task (Forgas et al., 1990).

Other tasks which have been used to provide false feedback to participants include: solving anagrams (Egloff, 1998; Stemmler, 1989), intelligence tests and social perception tasks (Ingram, 1984), concept formation tasks (McFarland & Ross, 1982; Baucom & Aiken, 1981), and computerised war games (Isen et al., 1978). However, although the technique has been widely implemented, there have been some concerns over the methodological detail and results have been equivocal.

One of the most quoted examples of the FF-MIP, a study on mood and memory by Isen et al. (1978), is unfortunately the most flawed. To manipulate mood, participants attempted to successfully complete a mission in a computerised war game. During the positive treatment participants the game was programmed to allow completion while in the negative treatment the game was programmed in such a way that successful completion was not possible (Isen et al., 1978). However, no mood assessments were administered during the experiment, therefore all conclusions were based purely on the assumption that the mood change would be congruent to the treatment valency.

Fortunately, other studies used appropriate methods of mood assessment. For example, Egloff (1998) showed that failure decreased positive affect and increased negative affect while success increased positive affect and decreased negative affect. However, as this study did not include a control condition, the only conclusion that can be drawn is that the success and failure conditions produce different moods. Unless the moods after these two

conditions were compared to that induced by a control condition, no inferences can be made as to the valency of the induced mood.

This problem is illustrated by a study by Baucom & Aiken (1981), who used the Depression Adjective CheckList (DACL; Lubin, 1965) to check the effect of the induction procedure. As there was a significant difference between the mean DACL score after the success treatment and failure treatment, the induction of a positive mood after success and negative mood after failure was concluded (Baucom & Aiken, 1981). However, that conclusion is less convincing when the mean DACL score after the success condition is compared to the normative data for non-depressed adults. During their validation of the DACL scale, Levitt and Lubin (1975) found that the mean DACL score in a population of non-depressed adults was 7.74 (Levitt & Lubin, 1975), while the mean DACL score after the success condition was 7.18 (Baucom & Aiken, 1981). Therefore, it appears that the success condition actually induced a neutral mood rather than a positive mood. As no comparison was made to a neutral condition, it was incorrectly assumed that because the mean DACL score fell into the depressive range the success condition produced the opposite effect, namely elation.

It does appear however that failure has more of an effect on mood than success. In a study where a control comparison was used, only the failure condition produced mood scores which were significantly different from the control condition (Ingram, 1984). So why should there be a discrepancy between the effects of success and failure? One possible explanation is the participant's attitude to performing the task on which the false feedback is given. It is likely that the majority of participants will endeavour to be successful, therefore learning of success will simply confirm their expectations and will have little

impact on their emotion. However, if their expectations were contravened by failure, it would be expected to be accompanied by a stronger emotional reaction (Ingram, 1984).

Another consideration is the complexity of the emotional reaction to success and failure, as the cognitive component is likely to be substantial (Forgas et al., 1990). The emotional consequences of success and failure described by Weiner et al (1979; 1978) can evolve into the more complex 'attribution-related affects' once the individual attempts to understand the reason behind their performance (McFarland & Ross, 1982). This more complex emotional outcome arises from the interaction between the performance outcome and the individual's locus of control. A person may feel positive if they attribute their success to be a result of their ability (internal) or if they feel their failure was a consequence of task difficulty (external), or vice versa (McFarland & Ross, 1982). In particular, McFarland & Ross (1982) found success was only associated with an increase in positive affect and decrease in negative affect when the subject's attributions around success were also manipulated.

The only constraint to choosing a suitable task to be used in the FF-MIP is that the use of feedback appears appropriate to the participant. As this means almost any task can be adapted for use, the most important consideration whether the task can serve more than one purpose. As working memory tasks play a prominent role in the theoretical basis of this thesis, an n-back working memory task has been chosen as this can easily be modified to include feedback. Also, as the task is intrinsically difficult, it is more likely that participants will attribute their success internally (McFarland & Ross, 1982), therefore increasing the likelihood of inducing a positive mood, which has been shown to be more difficult to induce than failure related negative moods (Ingram, 1984).



#### **4.1.3 Autobiographical Recall Mood Induction Procedure (ABR-MIP)**

The autobiographical recall mood induction procedure (ABR-MIP) is another example of a technique which originated in clinical practice and also one which places a strong emphasis on the role of cognition in emotional experiences. During his therapeutic sessions, Mosak would instruct his patients to use cognitive imagery to relive sad or happy events from past personal experience (Mosak & Dreikurs, 1973). Mosak would then use the situation to point out that there was a relationship between the way an individual thinks and feels about situations and experiences.

Taking this basic principle, Brewer, Doughtie & Lubin (1980) were the first to develop an autobiographical recall mood induction technique. Later adapted by Baker & Guttfreund (1993), this technique has a happy (ABR-happy), sad (ABR-sad) and neutral (ABR-neutral) condition. During the ABR-happy condition, the mood is induced by asking the participant to recall two happy events which made them feel as if they had everything going for them. The ABR-sad condition focused on events which made the participant feel lonely, rejected, defeated or hurt, while during the ABR-neutral condition the participant read a magazine article (Baker & Guttfreund, 1993). All conditions were followed by three questions so that the participant would remain focused on the imagination or reading task (Baker & Guttfreund, 1993).

There has been little investigation into the characteristics of the ABR-MIP since its development. The only change which has occurred is centred on the procedural detail neutral condition of the procedure. A number of experimenters (Wood et al., 1990; Salovy & Birnbaum, 1989) have adopted the procedure outlined by Wright & Mischel (1982),

where the same instructions are given during the happy, sad and neutral conditions. As a result, participants are instructed to imagine happy/sad/neutral (neither happy nor sad) events and are encouraged to feel the same happy/sad/neutral feelings they associate with the appropriate event (Wright & Mischel, 1982).

There is a risk during the procedure of Wright & Mischel (1982) that the participant would find it difficult to decide what constitutes a neutral event and feeling. As a result, this study will use the procedure outlined by Baker & Guttfreund (1993).

#### **4.1.4 Aims and predictions**

The purpose of this study is three-fold: (1) to test the effectiveness of the individual mood induction procedures, (2) to compare the three mood induction procedures to establish the most appropriate for use in the main studies of the thesis, and (3) establish whether heart rate can be used as an objective measure of mood.

The Positive Affect and Negative Affect Schedule scales (PANAS; Watson et al., 1988) will be used to measure the effectiveness of the manipulation procedures. This scale measures both a Positive Affect (PA) score and Negative Affect (NA) score, PA reflects enthusiasm and alertness, therefore a high PA score represents a state of high energy and pleasurable engagement while a low PA score is associated with sadness and lethargy (Watson et al., 1988). NA encompasses a wider feeling of distress and unpleasurable engagement, therefore a high score reflects a state of torment, contempt and nervousness while a low score is representative of serenity and calmness (Watson et al., 1988).

#### 4.1.4.1 Hypothesis 1

There is a clear picture of how a successful mood induction technique should alter scores on the PANAS scale. Regardless of the procedure, each treatment should be associated with the following mood profiles:

- elation treatments should increase PA scores and decrease NA scores;
- depression treatments should decrease PA scores have no effect on NA scores;
- neutral treatments should have no effect on either PA or NA scores.

Furthermore within each procedure, the neutral mood acts as a control state for the affective experience associated with the elation and depressed treatments. Consequently a specific pattern of changes in PA and NA scores are expected when the elation and depressed treatments are compared to the neutral treatment. Therefore, a successful elation induction should be associated with the following comparisons:

- the increase in PA score should be greater within the elation treatment than within the neutral treatment;
- the decrease in NA score should be greater within the elation treatment than within the neutral treatment.

Alternatively, a successful depressed induction should be associated with the following comparisons:

- the decrease in PA score should be greater within the depressed condition than within the neutral treatment;
- the changes in NA score within the depressed and neutral conditions should be comparably negligible.

#### **4.1.4.2 Hypothesis 2**

There has been no previous direct comparison of the False Feedback, Velten and Autobiographical Recall induction procedures. Consequently, as they are all capable of induction reflective moods there are no predictions as to which will be the most effective. However, it is predicted that the more robust procedure will be associated with greater changes in PA and NA scores.

#### **4.1.4.3 Hypothesis 3**

As the use of an objective measure alongside a self-report scale is preferable in mood induction studies, the use of a physiological correlate as a measure of mood was also investigated. Due to its close relationship with emotion and the ease with which it can be measured, heart rate was chosen as an appropriate physiological parameter. To establish the efficacy of heart rate as a physiological measurement of mood, baseline measures of heart rate will be compared to the induction procedure associated heart rate. It is predicted that the induction procedures will be associated with a change in heart rate, however as the evidence is equivocal the direction of change will not be predicted.

### **4.2 Method**

#### **4.2.1 Participants**

Ninety participants took part in this study, 45 female and 45 male, with an average age of 23.6 years. All participants gave signed consent prior to taking part in the study and they were paid for their participation. All participants had achieved a score of 9 or less on the Beck Depression Inventory (BDI; Beck, 1987). Four participants did not pass the screening procedure for the study as they scored more than 9 on the BDI. As this represents mild to moderate depression (Beck, 1987), it was deemed inappropriate to include them in a mood

manipulation study. All participants who failed to pass the selection criteria fully debriefed and informed that as their BDI score indicated they were feeling mildly unhappy it would be inappropriate to manipulate their mood.

#### **4.2.2 Measures**

Two measures were used to assess mood levels during this study, the Beck Depression Inventory (BDI; Beck, 1987) and the Positive Affect and Negative Affect Schedule scale (PANAS; Watson et al., 1988).

The BDI consists of 21 symptoms and attitudes, subdivided into four or five evaluative statements, which are designed to assess the severity of depression in adolescents and adults (Beck, 1987). It is widely accepted that the BDI is able to detect possible depression in normal populations (Steer et al., 1985), therefore it has been used to screen participants prior to undergoing the mood induction procedure.

The PANAS consists of 20 adjectives which are designed to produce measures of Positive Affect (PA) and Negative Affect (NA) (Watson et al., 1988). The scales have been shown to be a reliable, valid and efficient method of assessing PA and NA (Watson et al., 1988).

#### **4.2.3 Equipment**

A Grass amplifier was used to record the participants heart rate which was measured via a chest belt. A RM PC-5200 Professional Multimedia computer, with a 15-inch screen, was used to run the experimental sessions. Each experimental session was created in a Microsoft PowerPoint slideshow to allow consistency between participants.

#### **4.2.4 Mood Induction Procedures**

##### **4.2.4.1 The Velten Mood Induction Procedure**

Participants were told they were taking part in an investigation of suggestibility. Following Velten's (1967) instructions, participants were told that some psychologists believe that when people take tests, for example personality tests, they become influenced by the tests themselves. It was explained that by a process of self-suggestion, the individual's mood and thinking of themselves are swayed in the direction of the statements which make up the test. The participants were informed that the current study was investigating how strong this process of self-suggestion was.

After baseline recordings, the participants were prompted to read through an instruction booklet. The booklet instructed the participants to read through, in their heads and out loud, a series of statements which were about to be presented to them (Appendix 2). Participants were asked to imagine the statements represented their own thoughts, and were told that if they did not wish to continue with the experiment they were free to leave at any time. The instructions were adapted from those used by Velten (1967).

The computer prompted the participants to move from page to page as they read through the instruction booklet in order to maintain control over the pace of the experiment. The participants were then reminded that they should read each statement out loud and were instructed that the next slide would begin the series of statements. The participants were then presented with the appropriate procedure: the elation, depression or neutral treatment series (Appendix 2). The statements were taken from Velten (1967), however alterations were made to those statements which made particular reference to events specific to the

1960s or used American slang. Each series contained 60 statements, and individual statements were presented to participants for 10 seconds. A one second interval occurred between statements, and the presentation of a complete series took approximately 10 minutes.

#### 4.2.4.2 False Feedback Mood Induction Procedure

Participants were told they were taking part in an investigation of how learning a new task is influenced by regular feedback. They were told they would be performing a verbal working memory task referred to as a 3-back task, where each letter in a series would be compared to the letter which appeared three trials previously in the series.

Prior to the experimental session, the experimenter explained the nature of the task to the participant (Appendix 3). The participants were told that they would be presented with a series of letters on a background which consisted of a montage of letters. They were told that the letters could be in any position and could be presented in both upper- and lower-case. The participants were instructed that they should respond to each letter with a key press. If the letter was the same as that which appeared three trials previously they should press the 's' key and if the letter was different to that three trials previously they should press the 'd' key. All participants were asked to respond both quickly and accurately. They were reassured that the task was demanding and that it is common to feel that you are performing worse than you actually are. To ensure that participants understood the nature of the task, they were presented with a practice task on a sheet of paper. The experimenter remained with the participant until they were sure that they understood the task.

The experimenter then left the cubicle and started the experimental session. After the baseline recordings, the participants were reminded of the task instructions. They were then told the task would last 10 minutes and they would be presented with performance related information every two minutes.

The performance related information was automatically presented to the participant, regardless of their actual ability to do the task. During the elation treatment the participant was presented with information which conveyed the message that they were performing above average on the task. During the depression treatment, the participant was presented with information which conveyed the message that they were performing below average on the task. While during the neutral treatment, the participants were presented with basic information on the 3-back task they were performing. The feedback given to the participant in each of the conditions is presented in Appendix 3.

#### 4.2.4.3 Autobiographical Recall Mood Induction Procedure

Participants were told they were taking part in an investigation looking at imagination, in particular whether imagined scenarios which were externally or internally controlled differ. Following the baseline recordings, participants were given the treatment specific instructions, which were based on Baker & Guttfreund (1993).

During the elation treatment, participants were instructed to think of the two happiest events in their lives (Appendix 4). Participants were asked to spend 10 minutes thinking of events which made them feel on top of the world and that they had everything going for them. They were encouraged to think about how old they were, who else was involved and



what their feelings were. Participants were also instructed to sit back, close their eyes and relax whilst imagining the events. They were told that a beep would start and finish the imagination period, and they would be asked some questions about the events.

During the depression treatment participants were given the same instructions, but were asked to focus on the two saddest events in their lives (Appendix 4). The participants were encouraged to spend the 10 minutes thinking of the two events which made them feel hurt, rejected, defeated or lonely. Again, participants were asked questions at the end of the imagination period.

During the neutral treatment, participants were given an article on art museums (Perl, 2000) to read. They were instructed that after 10 minutes of reading time, they would be asked a number of questions related to the article. The questions were: 1] Did you find the article interesting? 2] Do you think it was well written and 3] Did you learn anything from this article?

In all treatments, the questions were asked to increase the participant's involvement in the imagination/reading task (Baker & Guttfreund, 1993).

#### **4.2.5 Design**

The study followed a 3x3 mixed factorial design. There were two between group independent variables, procedure (Velten, False Feedback, Autobiographical Recall) and valency (Elation, Depression, Neutral), and three within group dependent variables, PA score (pre-induction, post-induction), NA score (pre-induction, post-induction), and heart rate (baseline, induction period). Participants were randomly assigned to one of nine

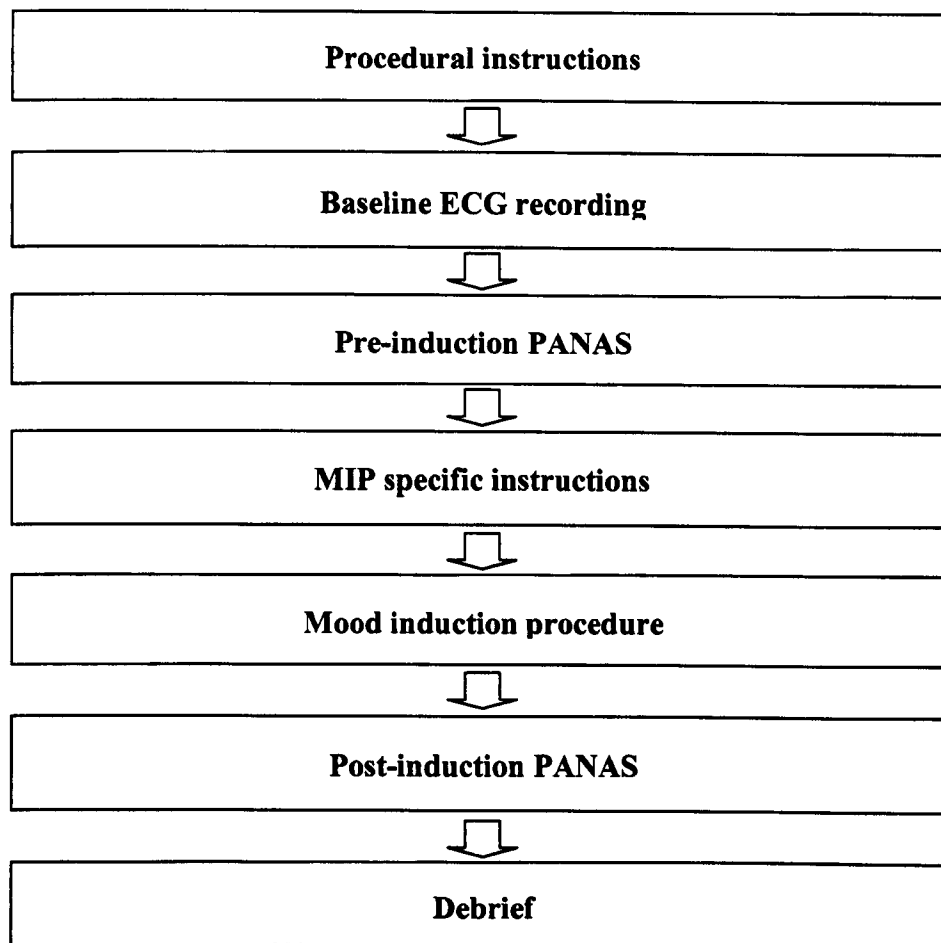
conditions: Velten Elation (E-VMIP), Velten Depression (D-VMIP), Velten Neutral (N-VMIP), False Feedback Elation (E-FF), False Feedback Depression (D-FF), False Feedback Neutral (N-FF), Autobiographical Recall Elation (E-ABR), Autobiographical Recall Depression (D-ABR), Autobiographical Recall Neutral (N-ABR). Each experimental group contained 10 participants and allocation to conditions was performed under the constraint that there was an equal number of males and females.

#### **4.2.6 Procedure**

Participants responded to a poster asking for paid volunteers to take part in an experiment looking at the effects of various psychological tests on the heart rate. Once the BDI had been completed and checked, a date for the experimental session was booked. This ensured that the BDI and the mood induction did not occur on the same day.

On entering the laboratory for the experimental session, the participant was told the cover story appropriate to their experimental condition. Each participant was asked to attach the chest belt underneath their clothing within the privacy of a curtained off section of the laboratory. The participant then entered the experimental cubicle and the remainder of the session was controlled by computer. The experimenter was able to see the participant through a one-way mirror at all times and the participant was reminded that they could stop the experiment at any time.

Each experimental session followed a generic procedure which was presented to the subject via a PowerPoint slide show (Figure 4.1). Participants then asked to fill out 'Form 1' in their participant booklet, this was the pre-induction PANAS. Next the participant was



**Figure 4.1** The generic experimental procedure followed in the PowerPoint slideshow used in all experimental conditions.

presented with the induction procedure specific instructions and they completed the induction procedure associated with their experimental condition (either E-VMIP, D-VMIP, N-VMIP, E-FF, D-FF, N-FF, E-ABR, D-ABR, or N-ABR). Immediately after, they were instructed to complete ‘Form 2’ in the participant booklet, the post-induction PANAS. The participant was then asked to remain seated until the experimenter entered the cubicle. The experimenter then unattached the chest belt from the Grass recorder and fully debriefed the participant on the nature of the experiment.

Where the participant had completed a depressive treatment, they were shown the ‘Cat drilling’ stand up routine by Eddie Izzard (Izzard, 1996) in order to return their mood to baseline. The participant was then left alone to remove the chest belt. Once the participant left the cubicle they were thanked and paid for their participation.

#### **4.2.7 Data Reduction**

##### **4.2.7.1 PANAS scores**

Change scores (pre-induction score minus post-induction score) were computed to index changes in mood profiles associated with each induction procedure for positive affect scores (PADIF) and negative affect scores (NADIF). A negative change score reflects an increase on the PANAS mood scale and a positive change score reflects a decrease on the PANAS mood scale.

##### **4.2.7.2 Heart rate measures**

The baseline value of heart rate was recorded at the beginning of the experimental session. The mean induction value of heart rate was computed from three recordings during the induction procedure (first minute; middle minute, final minute) (Waldstein et al., 2000). Change scores (baseline value minus mean induction value) were calculated to index the changes in heart rate associated with each induction procedure (HRDIF).

#### **4.2.8 Statistical Analysis**

A separate two-way ANOVA analysis was run on the change scores calculated for each of the dependent variables. Therefore the change in positive affect scores (PADIF), the change in negative affect scores (NADIF) and change in heart rate (HRDIF) were analysed independently. Within each of these analyses, mood induction procedure (False Feedback,

Velten, Autobiographical Recall) and valency (Elation, Neutral, Depressed) were included as factors. Where significant effects were found, the relationship between the levels of the experimental conditions was investigated using a Bonferroni corrected pairwise comparison.

## 4.3 Results

### 4.3.1 Descriptive Statistics

The means and standard deviations for the change scores associated with the three dependent variables (PADIF, NADIF, HRDIF) are presented in Table 4.1.

Induction Procedure	Valency	PADIF		NADIF		HRDIF	
		Mean	SD	Mean	SD	Mean	SD
False feedback	Elation	1.3	2.7	0.8	5.7	-6.5	4.5
	Neutral	2.5	5.2	-4.1	8.5	-3.5	5.5
	Depressed	4.2	5.3	-2.9	7.5	-7.2	4.2
Velten	Elation	-2.1	5.6	1.0	2.2	-3.3	5.8
	Neutral	1.8	7.6	2.5	4.5	-3.3	4.4
	Depressed	6.2	7.7	-1.7	4.5	-4.0	3.2
Autobiographical recall	Elation	-0.1	3.2	-0.1	6.2	-4.7	2.8
	Neutral	3.9	4.4	0.0	2.7	-2.9	4.1
	Depressed	5.4	8.8	-1.90	3.1	1.2	5.0

**Table 4.1** The mean changes and standard deviations for PADIF (change in positive affect), NADIF (change in negative affect) and HRDIF (change in heart rate).

To assess the stability of the baseline mood assessment, a comparison was made to the original normative data to assess whether the scores were representative of a normal population (Table 4.2). An independent t-test, using pooled variance as the sample sizes

were unequal, was applied to the PA score and NA scores. For the PA score,  $t = 1.27$  ( $p = > 0.05$ ) and for the NA scores  $t = -0.30$  ( $p = > 0.05$ ). In both cases there is no significant difference between the study score and the normative data, therefore it can be assumed that the baseline data is representative of a normal population.

Subscale	Normative data (Watson et al, 1988) N = 660		Current study N = 90	
	Mean	SD	Mean	SD
PA	29.7	7.9	28.58	6.6
NA	14.8	5.4	14.99	5.16

**Table 4.2** Normative PANAS data (Watson et al, 1988) in comparison with study baseline data.

#### 4.3.2 Positive Affect (PADIF)

There was no significant effect of mood induction procedure ( $F(2,81) = 0.264$ ,  $p = > 0.05$ ), therefore the change in positive affect associated with each induction procedure did not differ. The estimated group means and standard errors are presented in Table 4.3.

Induction Procedure	Mean	SE
False Feedback	2.67	1.08
Velten	1.96	1.08
Autobiographical Recall	3.067	1.08

**Table 4.3** Mean change in positive affect and standard errors (SE) calculated for the three mood induction procedures.

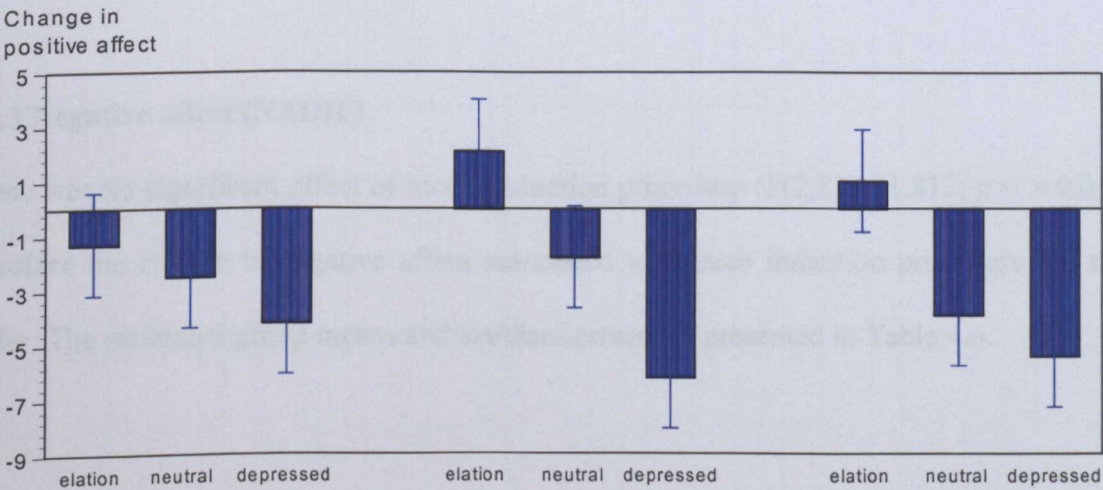
There was a significant main effect of **valency** ( $F(2,81) = 6.627$ ,  $p = 0.002$ ), the estimated group means and standard errors for which are presented in Table 4.4. The elation treatment is associated with a small increase in positive affect and the depressed treatment is

associated with a decrease in positive affect. A pairwise comparison showed that the mean change associated with these treatments was significant ( $p = 0.01$ ).

Valency	Mean	SE
Elation	-0.30	1.08
Neutral	2.73	1.08
Depressed	5.267	1.08

**Table 4.4** Mean change in positive affect and standard errors (SE) calculated for the three valency treatments associated with the mood induction techniques.

The interaction between mood induction procedure and valency was not significant ( $F(4,81) = 0.59, p = > 0.05$ ) (Figure 4.2). During the False Feedback condition a decrease in positive affect was associated with each of the valency treatments. Within the Velten and Autobiographical Recall procedures the expected change was seen in the elation and depressed treatments, an increase in positive affect during the elation treatment and a decrease in positive affect during the depressed treatments.



**Figure 4.2** Mean change in positive affect recorded for the three valency treatments within each mood induction technique. A positive change is associated with an increase, and a negative change is associated with a decrease in positive affect.

#### 4.3.2.1 Within-procedure comparisons

The pairwise comparison between changes in PA scores between elation-neutral treatments and depressed-neutral treatments for each of the mood induction procedures are presented in Table 4.5. Only the depressed treatment of the Velten mood induction procedure produced the required significant pairwise comparisons.

Induction Procedure	Pairwise comparison	Significance
False feedback	Elation vs Neutral	Non significant
	Depressed vs Neutral	Non significant
Velten	Elation vs Neutral	Non significant
	Depressed vs Neutral	p = 0.05
Autobiographical recall	Elation vs Neutral	Non significant
	Depressed vs Neutral	Non significant

**Table 4.5** Pairwise comparisons for changes in PA scores between treatments for each of the mood induction procedures.

#### **4.3.3 Negative affect (NADIF)**

There was no significant effect of mood induction procedure ( $F(2,81) = 1.817$ ,  $p = > 0.05$ ), therefore the change in negative affect associated with each induction procedure did not differ. The estimated group means and standard errors are presented in Table 4.6.



Induction Procedure	Mean	SE
False Feedback	-2.067	0.9
Velten	0.60	0.9
Autobiographical Recall	-0.667	0.9

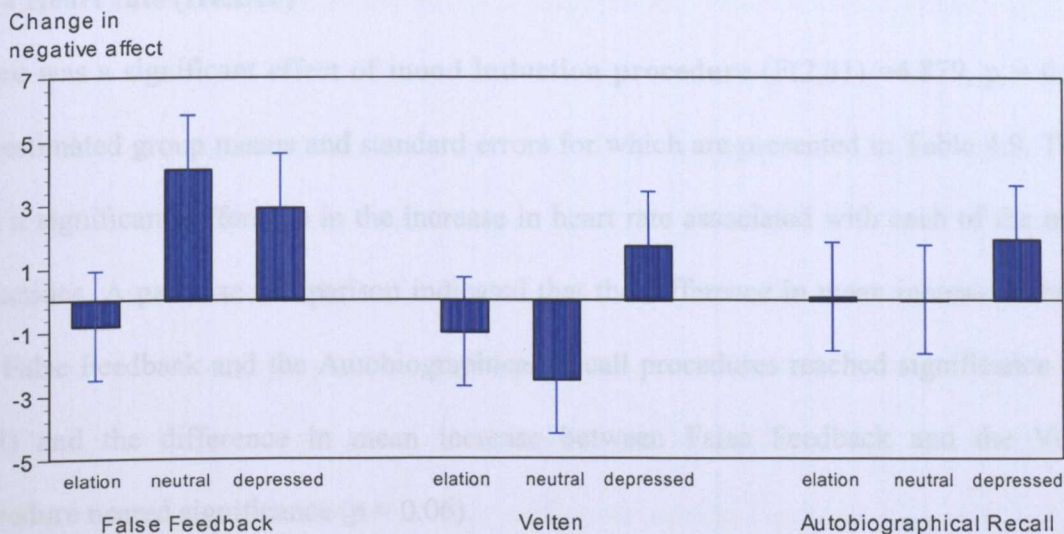
**Table 4.6** Mean change in negative affect and standard errors (SE) calculated for the three mood induction procedures.

There was no significant effect of valency ( $F(2,81) = 1.931, p = > 0.05$ ), therefore the change in negative affect associated with each treatment valency did not differ. The estimated group means and standard errors for which are presented in Table 4.7.

Valency	Mean	SE
Elation	0.567	0.9
Neutral	-0.533	0.9
Depressed	-2.167	0.9

**Table 4.7** Mean change in negative affect and standard errors (SE) calculated for the three valency treatments associated with the mood induction techniques.

The interaction between mood induction procedure and valency was not significant ( $F(4,81) = 1.110, p = > 0.05$ ) (Figure 4.3). The change in negative affect seen was not consistent with all of the expected treatment profiles in any of the mood induction procedures.



**Figure 4.3** Mean change in negative affect recorded for the three valency treatments within each mood induction technique. A positive change score is associated with an increase in negative affect and a negative change score is associated with a decrease in negative affect.

#### 4.3.3.1 Within-Procedure Comparisons

The pairwise comparison between changes in NA scores between elation-neutral treatments and depressed-neutral treatments for each of the mood induction procedures are presented in Table 4.8. None of the treatments produced the required significant pairwise comparisons.

Induction Procedure	Pairwise comparison	Significance
False feedback	Elation vs Neutral	Non significant
	Depressed vs Neutral	Non significant
Velten	Elation vs Neutral	Non significant
	Depressed vs Neutral	Non significant
Autobiographical recall	Elation vs Neutral	Non significant
	Depressed vs Neutral	Non significant

**Table 4.8** Pairwise comparisons for changes in NA scores between treatments for each of the mood induction procedures.

4.3.4 Heart rate (HRDIF)

There was a significant effect of **mood induction procedure** ( $F(2,81) = 4.879, p = 0.01$ ), the estimated group means and standard errors for which are presented in Table 4.9. There was a significant difference in the increase in heart rate associated with each of the mood inductions. A pairwise comparison indicated that the difference in mean increase between the False Feedback and the Autobiographical Recall procedures reached significance ( $p = 0.03$ ) and the difference in mean increase between False Feedback and the Velten procedure neared significance ( $p = 0.06$ ).

Induction Procedure	Mean	SE
False Feedback	-5.76	0.83
Velten	-3.53	0.83
Autobiographical Recall	-2.13	0.83

**Table 4.9** Mean change in heart rate and standard errors (SE) calculated for the three mood induction procedures.

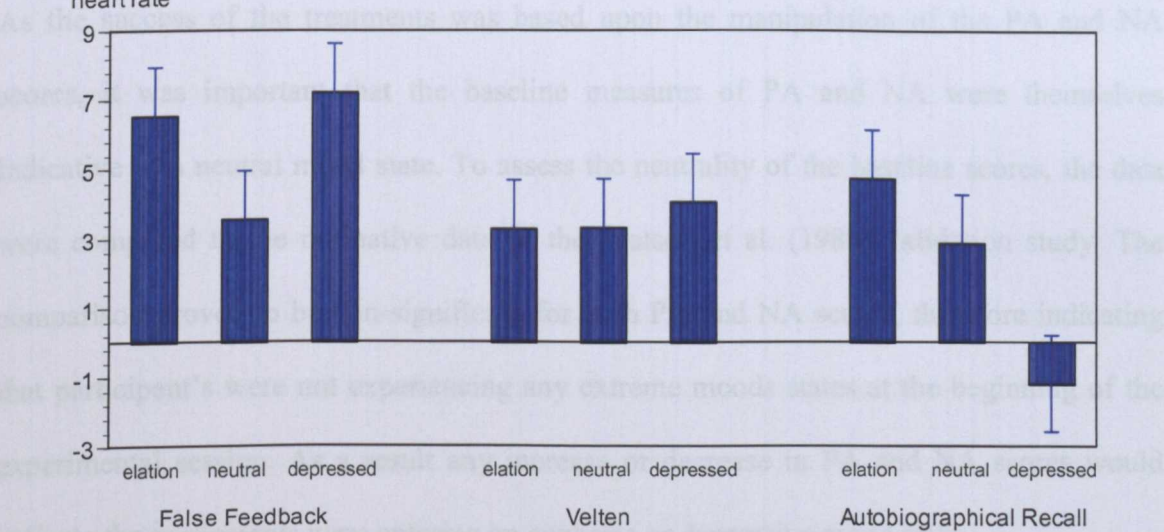
There was no significant effect of valency ( $F(2,81) = 1.184, p = > 0.05$ ). Each treatment was associated with an increase in heart rate, however this increase did not differ with the treatment valency. The estimated group means and standard errors for which are presented in Table 4.10.

Valency	Mean	SE
Elation	-4.84	0.83
Neutral	-3.23	0.83
Depressed	-3.34	0.83

**Table 4.10** Mean change in heart rate and standard errors (SE) calculated for the three valency treatments associated with the mood induction techniques.

The interaction between **mood induction procedure** and **valency** was significant ( $F(4,81) = 2.629, p = 0.04$ ) (Figure 4.4). An increase in heart rate was associated with all conditions apart from the depressed treatment within the Autobiographical Recall procedure. There were larger increases in heart rate within the False Feedback condition. A pairwise comparison revealed that the change in heart rate within the autobiographical recall depressed treatment was significantly different to the change associated with both the false feedback elation ( $p = 0.05$ ) and depressed ( $p = 0.05$ ) treatments. The group estimated means and standard deviations for this interaction are presented in Table 4.1.

no affect upon PA and NA scores.



**Figure 4.4** Mean change in heart rate recorded for the three valency treatments within each mood induction technique. A positive change score is associated with an increase in heart rate and a negative change score is associated with a decrease in heart rate.

There was a significant main effect of valency ( $p = 0.003$ ) which reflected that the change associated with each of the treatment differed. As predicted the elation

#### 4.4 Discussion

##### 4.4.1 Efficacy of Mood Induction Procedures

Determining the efficacy of the three mood induction procedures involved two stages. Firstly, the profile of the mood state induced by each of the treatments (elation, depressed

and neutral) was assessed. Secondly a comparison was made between the affective states (elation, depressed) and the control neutral mood state.

#### 4.4.1.1 Mood State Profiles

Each of the affective states induced by the induction procedures under investigation is associated with a specific mood profile. A successful elation treatment should increase PA scores and decrease NA scores, a successful depression treatment should decrease PA scores and have no effect upon NA scores, while a successful neutral treatment should have no effect upon PA and NA scores.

As the success of the treatments was based upon the manipulation of the PA and NA scores, it was important that the baseline measures of PA and NA were themselves indicative of a neutral mood state. To assess the neutrality of the baseline scores, the data were compared to the normative data of the Watson et al. (1988) validation study. The comparison proved to be non-significant for both PA and NA scores, therefore indicating that participant's were not experiencing any extreme moods states at the beginning of the experimental session. As a result any increase or decrease in PA and NA scores would indicate the participants were entering an euphoric or depressive mood state.

##### *4.4.1.1.1 Positive affect*

There was a significant main effect of valency ( $p = 0.002$ ) which indicated that the change in positive affect associated with each of the treatment differed. As predicted the elation treatment were associated with an increase in positive affect, albeit small, and the depression treatment was associated with a decrease in positive affect. The neutral treatment was associated with a small decrease in positive affect.



However, it can be seen in Figure 4.2 that the expected mood state profile was not associated with each of the mood induction procedures. The least effective was the False Feedback induction procedure, where each of the valency treatments was associated with a reduction in PA scores. Therefore, regardless of whether the feedback given to the participant was positive, negative or neutral, the participant experienced a decrease in positive affect.

In relation to the elation and depressed treatments, both the Velten and Autobiographical Recall procedures were associated with correct mood profile. Therefore reading the elation Velten statements and remembering positive life events led to an increase in positive affect, while reading the depressive Velten statements and remembering negative life events led to a decrease in positive affect.

Unfortunately, both the neutral treatments also led to a decrease in positive affect. When the tasks involved in the neutral treatments are considered, it is hardly surprising that there was some degree of negative impact upon the participant's positive mood. During the creation of a non-emotive task it is very difficult to achieve the fine balance between emotionless and boring. The neutral Velten statements outlined in Appendix 2, such as "Agricultural products comprised seventy percent of the income" and "Utah is known as the beehive state" can be described as tedious.

Similarly, the magazine article employed in the neutral Autobiographical Recall treatment had to be uninteresting in order for it to remain emotionless. Therefore in both cases, the critical factor will be the suitability of the neutral mood as a control condition. Consequently the degree of change associated with the neutral treatment and the

comparison of this change with the affective treatments will indicate the effectiveness of these two procedures.

#### *4.4.1.1.2 Negative affect*

There was no significant main effect of valency associated with the change in negative affect, indicating that a similar degree of change in negative affect was associated with the elation, depressed and neutral treatments. As the mood profile for the neutral and depressed treatments is no change in negative affect, the actual magnitude of change will be the most important consideration here. In all treatments, a small change in mood was recorded.

It can be seen in Figure 4.3 that the only procedure treatment to fulfil its criteria is the Autobiographical Recall neutral treatment, where no change in negative affect was seen across the induction period. Under all other treatments the inappropriate change in negative affect was recorded. Again, the comparison between the changes recorded over the affective and neutral states will provide the most information on the effectiveness of the procedures as it will be the magnitude of these changes that will be important.

#### *4.4.1.1.3 Comparison between neutral and affective state*

As the three treatments within each mood induction should produce three distinct mood states, the neutral treatment can act as a baseline to the elation and depression treatments. Therefore if the elation and depression treatments produce robust states of euphoria and depression, changes in PA and NA, where appropriate, should be greater than during the neutral treatment.

In terms of the positive treatments, none of the induction procedures produced an appropriate change in positive or negative affect that was significantly larger than the change recorded over the neutral treatment. There are two possible explanations as to why an euphoric mood is difficult to induce. Firstly, as the normative data of the PANAS scale shows (Watson et al., 1988), the mood profile of a normal individual is associated with a reasonable high positive affect and a low negative affect. It is possible that positive treatments simply reinforce the individual's existing mood state rather than raise them into a state of euphoria.

Secondly, the use of a global treatment may not be suitable for euphoric moods. The things in life which make people feel extremely happy tend to be specific to the individual. It is possible that the only way an euphoric mood can be induced is by using an individually tailored technique. In this way the things which make someone happy, for example specific pieces of music or film clips, would be chosen prior to the mood induction therefore ensuring that individual has a psychological connection with the treatment.

In terms of the depressed treatments, the only comparison which produced a mean difference in change that was significantly different was between the depressed and neutral treatments of the Velten induction procedure ( $p = 0.05$ ). Therefore the decrease in positive affect produced by depressed treatment is significantly larger than the decrease associated with neutral treatment. This is in line with individual experiencing boredom during the neutral treatment and therefore experiencing a small reduction in positive affect, while the depression treatment results in a more considerable reduction in positive feelings.



In all other cases, the mood states created by the other treatments were not sufficiently distinct. Even where the change in mood state fitted the expected profile, for example the depressed treatment of the Autobiographical Recall procedure, the neutral treatment also induced a change in mood. This highlights the difficulty of creating a neutral induction procedure that is emotionless yet not so dull as to act as an inducer of negative mood.

Therefore, this reinforces the bias within affective neuroscience research that the majority of studies investigate the influence of negative moods upon cognitive performance. Although the Resource Allocation Model makes predictions on the influence of positive mood upon performance, the difficulty surrounding the production of a true euphoric mood state means the focus will remain upon depressed moods.

#### **4.4.2 Comparison of Mood Induction Procedures**

The lack of significant effect of induction procedure, in relation to both positive and negative affect, means that the magnitude of change did not differ across the induction procedures. Therefore there is no one induction procedure which stands out as being the most effective at manipulating mood. Therefore the decision on which mood induction procedure should be used in this thesis must be based upon the mood profiles generated and the quality of the difference between neutral and depressed moods.

##### **4.4.2.1 The False Feedback Procedure**

The false feedback procedure gave rise to neutral and depressed mood profiles that were both inappropriate and identical, as both treatments were associated with a decrease in positive affect and an increase in negative affect. This denotes that the participants shifted towards a state of sadness, nervousness and lethargy, regardless of what form of feedback

they received. It appears that performance of the working memory task, which formed the basis of the procedure, was sufficient to have a negative effect upon mood. Therefore the false feedback mood induction procedure, comprising of the 3-back working memory task as the basis for feedback, is not effective at inducing moods.

#### 4.4.2.2 Autobiographical recall

The autobiographical recall procedure achieved more appropriate mood profiles but the changes recorded were not of a sufficient magnitude. The influence on negative affect was as expected in both treatments with either small or no changes being recorded and no significant difference between the change during the neutral and depressed treatments. In relation to positive affect however, an increase was associated with both the neutral and depressed treatments. As a differential effect upon positive affect was expected between the two treatments, the non significant pairwise comparison meant that the neutral treatment did not act as a suitable control for the depressed treatment.

Considering the profile of the moods associated with this procedure it appears that the main problem is the neutral treatment. When an individual recalls sad memories, although not associated with a strong effect, there is a pure influence upon their emotions as their sadness increases without an associated increase in anxiety levels. It is apparent that simply reading an article is not a suitable neutral activity as this is associated with a comparable increase in sadness.

One possibility would be to ask individuals to remember events that had no emotional impact upon them, however it is likely that the individual would have to classify their memories in order to find a suitably emotionless memory. During the recollection events,

both positive and negative memories could be triggered which would be deemed unsuitable once the full circumstance of the memory is remembered. Therefore there would be no control upon the actual emotional experience of the individual. Until a suitable control treatment is developed, the autobiographical recall procedure is not effective at inducing moods.

#### 4.4.2.3 The Velten Mood Induction Procedure

The Velten statements produced the most effective depressed and neutral mood. Although there were small influences on negative affect during both treatments, there was no significant difference in the mean change over both conditions. Therefore levels of nervousness and anxiety were consistently low during the neutral and depressed moods. In relation to the impact on positive affect, this treatment was the only one to be associated with a significant pairwise comparison ( $p = 0.05$ ) when the mean changes were compared. Therefore the increase in sadness and lethargy experienced was greater in the depressed condition than the neutral condition.

The distinction between the mood profiles associated with the depressed and neutral Velten treatments means that this induction procedure will be used within this investigation of the influence of depressed mood upon cognitive processes.

#### **4.4.3 Heart Rate as a Psychophysiological Parameter of Mood**

There was a significant difference in the heart rate change which occurred across the three induction procedures ( $p = < 0.01$ ). Through a pairwise comparison it became apparent that the increase within the false feedback procedure was significantly greater than that within the autobiographical recall procedure ( $p = 0.05$ ). While the comparison of the increases

between the false feedback and Velten procedures neared significance. As the large increase in heart rate is associated with the false feedback procedure, it is likely that this reflects a heightened arousal caused by the feedback received and the performance of the inherently difficult 3-back working memory task.

The finding that there was no significant difference across the valency of the treatment supports the theory that emotion-related changes in heart rate reflect a generalised emotional or task-related arousal response (Waldstein et al., 2000; Nyklicek et al., 1997; Warner & Strowman, 1994; Cacioppo et al., 1993). Therefore heart rate is not a suitable psychophysiological, and therefore objective, measure of mood.

The significant interaction which occurred between mood induction procedure and valency does not alter this conclusion. It is clear from the pairwise comparisons that this interaction is a result of the large increases in heart rate associated in the affective treatments of the false feedback procedure and the small decrease in heart rate associated with the autobiographical recall depressed treatment. Therefore it again reflects the greater psychological arousal associated with the false feedback procedure.

#### **4.5 Conclusion**

The magnitude of change induced in mood profiles did not differ when false feedback, autobiographical recall and Velten mood induction procedures were compared. In regard to positive treatments, none of the techniques were able to create the appropriate change in mood profile, therefore reinforcing the decision that only depressed moods will be investigated in this thesis. Only the Velten mood induction procedure created changes to the neutral and depressed mood profiles that resulted in a clear differentiation between the

two mood states, therefore allowing the neutral treatment to act as a control for the depressed treatment. Therefore this thesis will utilise the Velten mood induction technique. Heart rate was shown to vary as a function of psychological arousal rather than affective state and therefore will not be used as an objective measure of mood.

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## **Chapter 5**

### **Mood and Cognition I – The influence of depressed mood on a passive attention task**

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#### **5.1 Introduction**

Information processing can be seen to vary along a continuum of cognitive intensity, which represents the number of cognitive sub-processes that must be completed in order to successfully accomplish task goals. The Resource Allocation Model (Ellis et al., 1988) postulates that depression will have a detrimental effect upon performance if the emotional experience leaves insufficient resources available for cognitive processing. This thesis proposes that cognitive tasks controlled by neuroanatomical regions also associated with the experience of depression will be more susceptible to the detrimental effects of depression, due to their dependence upon a common resource reserve.

Consequently performance on a low cognitive intensity task, such as stimulus identification, should not be altered by a depressed mood. By utilising electrophysiological techniques the neurological signature of cognitive processes can be compared, therefore providing a means of quantifying the temporal dynamics of information processing under a neutral and depressed mood. An oddball task will be employed in this investigation as it elicits the P300 component, which is thought to index stimulus identification (Polich et al., 1995).

### 5.1.1 The P300 Component

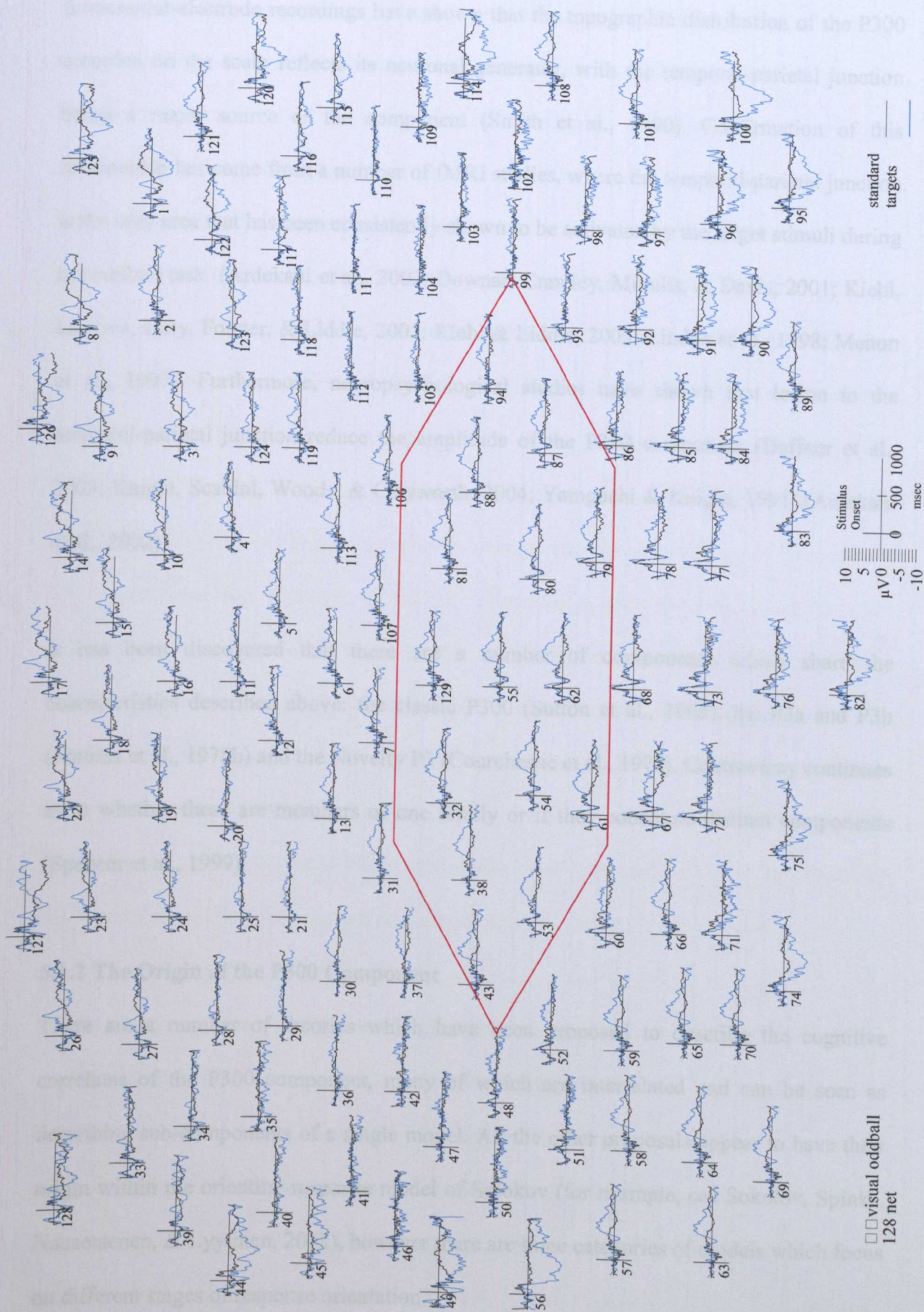
The P300 is an electrophysiological phenomenon associated with basic information processing mechanisms (Polich et al., 1995). The presence of this component within the ERP waveform is associated with number of different stimuli as the crucial factor in its elicitation is the relevance of the stimuli to the viewer, rather than the stimuli per se. Therefore, rather than being sensitive to the physical characteristics of the stimulus, the P300 is thought to be influenced by the information contained within the stimulus and the context of presentation (Squires et al., 1975a; Sutton et al., 1965).

The traditional method of elicitation is the oddball paradigm (Picton, 1992; Polich, 1999), which can be run as a visual or auditory task. The principle in both presentation modalities is the same; the participant is presented with a random series of standard and target stimuli at a ratio of 80:20. They are instructed to respond when they hear or see a target stimulus and to make no response when a standard stimulus is presented.

When event-related potentials are created for both categories of stimuli, a positive deflection, in the region of 10 to 20  $\mu\text{V}$ , at around 300ms post stimulus presentation is associated with the target stimuli in comparison to the standard stimuli (Polich et al., 1995). Figure 5.1 shows the event-related potentials achieved during a dense array recording of a classic oddball experiment<sup>2</sup>. It can be seen that the topographic distribution of this waveform component varies across the scalp, with maximum differentiation between standard and target stimuli occurring in the parietal region.

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<sup>2</sup> This data was recorded by the author during a pilot study to ensure that the newly installed dense array EEG system was working correctly.



**Figure 5.1** The grand averaged ERP waveforms from an oddball paradigm, the area within the red hexagon approximately represents the parietal region of interest



Intracranial-electrode recordings have shown that the topographic distribution of the P300 recorded on the scalp reflects its neuronal generator, with the temporal-parietal junction being a major source of the component (Smith et al., 1990). Confirmation of this relationship has come from a number of fMRI studies, where the temporal-parietal junction is the only area that has been consistently shown to be activated by the target stimuli during the oddball task (Ardekani et al., 2002; Downar, Crawley, Mikulis, & Davis, 2001; Kiehl, Laurens, Duty, Forster, & Liddle, 2001; Kiehl & Liddle, 2003; Linden et al., 1998; Menon et al., 1997). Furthermore, neuropsychological studies have shown that lesion to the temporal-parietal junction reduce the amplitude of the P300 component (Daffner et al., 2003; Knight, Scabini, Woods, & Clayworth, 2004; Yamguchi & Knight, 1991). (Ardekani et al., 2002)

It has been discovered that there are a number of components which share the characteristics described above: the classic P300 (Sutton et al., 1965), the P3a and P3b (Squires et al., 1975b) and the Novelty P3 (Courchesne et al., 1975). Controversy continues as to whether these are members of one family or if they constitute distinct components (Spencer et al., 1999).

### **5.1.2 The Origin of the P300 Component**

There are a number of theories which have been proposed to describe the cognitive correlates of the P300 component, many of which are interrelated and can be seen as describing sub-components of a single model. All the other proposals appear to have their origin within the orienting response model of Sokolov (for example, see Sokolov, Spinks, Naeaetaenen, & Lyytinen, 2002), however there are three categories of models which focus on different stages of response orientation.

First are those theories which focus upon environmental context appraisal. It is proposed that the P300 reflects an attentional system centered on the individual's relationship with the environment. Within this model, the P300 has been described as representing an external control system which focuses attentional mechanisms towards environmental features (Kok, 1990). In a similar vein, the component has been portrayed as demonstrating an attentional shift towards salient events in the environment which will influence the development of future cognitive strategies (Pritchard, 1981). In either case, the component is thought of as orienting the individual to information, contained within the stimulus or event, which will have future consequences for them.

The second category of theories assesses the task of context updating. It has been argued that when observing a series of stimuli or events, a individual is continually and automatically making predictions about the next stimulus or event they will encounter (Squires, Wickens, Squires, & Donchin, 1976). Consequently, it is thought that a P300 is elicited if a violation of the contextual hypothesis occurs, thereby representing an acknowledgement of the infringement and the revision of the individual's schemata (Squires et al., 1976). In line with this, it has also been suggested that the P300 represents the transfer of pertinent information into consciousness (Picton, 1992).

Finally, there are the theories which focus on the memory processes which underlie contextual revisions. For example, the P300 is thought to index the maintenance of information within working memory whilst the individual's model of stimulus context is updated (Donchin, Karis, Bashmore, Coles, & Gratton, 1986). Taking this a step further, the cellular activation which gives rise to the P300 component is thought to reflect the actual change in the neuronal representation of the stimulus (Polich, 1989).

The realisation that the P300 is actually a number of sub-components provides a means of combining these models to create a comprehensive account of the origins of this component. The activation associated with the P3a is more anterior and slightly earlier than is associated with the traditional parietal P300 deflection (Squires et al., 1975b). Therefore this frontal activity could account for the realisation that an event has violated the individual's contextual hypothesis. The later, more posterior activation could then represent the rebuilding of the neuronal representation of the new contextual information.

Less controversy surrounds the latency of the P300 component. The general consensus is that it reflects the time-point during information processing that stimulus evaluation occurs (Kutas, McCarthy, & Donchin, 1977).

### **5.1.3 The Stability of the P300 Component**

As previously stated, the P300 is not influenced by the physical characteristics of the stimulus. Instead factors such as: stimulus probability, stimulus significance, task difficulty, motivation and vigilance have all been shown to modulate the amplitude of the component (Johnson, 1986; Johnson, 1993; Sommer & Matt, 1990). Interestingly, the latency of the component has been shown to be independent of the behavioural reaction time (Duncan-Johnson, 1981; Verleger, 1997) and is therefore sensitive to the complexity of the task rather than the actual response selection process being initiated (Smulders, Kok, Kenemans, & Bashmore, 1995; McCarthy & Donchin, 1981).

In addition to these cognitive influences, there are a number of spontaneous or environmentally induced variations which may occur (Polich & Herbst, 2000). Table 5.1

provides an overview of the influence of these factors, which are collectively described by Polich & Kok (1995) as ‘biological determinants’.

Biological determinant	Amplitude	Latency
Natural		
Circadian rhythms	Indirect	Indirect
Body temperature	No	Yes
Heart rate	No	Yes
Food intake	Yes	Some
Induced		
Exercise	Indirect	Direct
Fatigue	Yes	Yes
Caffeine	Some	Yes
Nicotine	Small	Yes
Alcohol	Yes	Yes
Age	Yes	Yes
Intelligence	Yes	Yes
Gender	Small	Small
Personality	Yes	No

**Table 5.1** The biological determinants, as outlined by Polich and Kok (1995) and their influence upon the P300 ERP component. (Adapted from Polich & Herbst, 2000).

### 5.1.4 The P300 and Psychopathological Illness

#### 5.1.4.1 Schizophrenia

As a result of the experimental robustness associated with the P300 component, it has a history of being used as a biological marker during the assessment of psychopathological disorders. For example, in schizophrenia a common observation within patients suffering from the disorder is an amplitude reduction in response to auditory oddball stimuli (Pritchard, 1986; Regan, 1989).

Within patients with schizophrenia, a more controversial characteristic is the existence of hemispheric asymmetries (Hill & Weisbrod, 1999). Reports have been made of both the

presence of left hemisphere amplitude reductions (Morstyn, Duffy, & McCarley, 1983; McCarley, Faux, Shenton, Nestor, & Holinger, 2004; Faux et al., 1993; Strik, Dierks, Francek, Stoeber, & Maurer, 1994) and the absence of such asymmetries (Pfefferbaum, Ford, White, & Roth, 1989; Moore, Tucker, & Coburn, 1992; Ford et al., 1994). Alternatively there have been reports of the reverse situation, where the amplitude reduction is associated with the right hemisphere (Maurer & Dierks, 1987).

In an attempt to rectify this disparity, Hill & Weisbrod (1999) investigated the general characteristics of the P300 component, including asymmetry, in schizophrenia. Within the clinical population, there was a correlation between general amplitude and hemispheric asymmetry: left hemispheric amplitude reduction was associated with low general amplitude and right hemispheric reduction was associated with high general amplitude (Hill et al., 1999). When participants were reassessed on the basis of their amplitude characteristics rather than their diagnostic classification, it was found that left hemispheric asymmetry was only an indicator of psychopathology when coupled with low general amplitudes (Hill et al., 1999). However the existence of both directions of asymmetry within the patient and control populations led to the conclusion that these patterns of activity were not specific to schizophrenia.

#### 5.1.4.2 Depression

There is less work surrounding the findings within mood disorders, and there is even more controversy surrounding the effect of depression upon the P300 component. Evidence has been reported of a reduction in P300 amplitude (Diner et al., 1985; Gangadhar et al., 1993; Muir et al., 1991; Shagass et al., 1981), while other studies have shown no difference in amplitude between depressed patients and healthy controls (Bruder et al., 1991; El

Massioui et al., 1988; Giedke et al., 1980; Have et al., 1991; Plooij-Van Gorsel, 1984; Kaustio et al., 2002; Sara et al., 1994).

The same picture surrounds the characteristics of the latency of the P300 component. Prolonged latencies have been reported in depressed patients by a number of studies (Bruder et al., 1991; Vandoolaeghe, van Hunsel, Nuyten, & Maes, 1998; Himani, Tandon, & Bhatia, 1999), while others have shown no difference when depressed patients were compared to healthy controls (Kaustio et al., 2002).

One of the main reasons for this inconsistency is the diagnostic criterion applied to patients. It has been argued that the heterogeneity of depressed patients may actually suppress the presence of effects which are specific to a sub-population of patients (Hansenne et al., 2000). There are a number of affective, cognitive and somatic symptoms associated with the experience of depressive episodes (Akiskal, 2000), and as these may be linked to distinct neurobiological processes there is a possibility that each will have a differential effect upon the P300 (Kaustio et al., 2002).

In patients exhibiting impulsivity as the dominant symptom of their depressive episode, there is an increase in P300 amplitude when compared to patients presenting blunted affect (Pierson et al., 1991; Pierson et al., 1994; Pierson et al., 1996). Individuals experiencing paranoid symptoms during a major depressive episode with psychotic features have shown reduced P300 amplitudes in comparison to those patients not exhibiting psychotic symptoms (Karaaslanb, Gonul, Oguz, Erdinc, & Esel, 2003; Kaustio et al., 2002). When patients presenting affective symptoms were compared to those exhibiting psychotic symptoms within a depressive episode, there was an asymmetry in amplitude reduction

specific to right temporal region (Kaustio et al., 2002). Prolonged latency has only been attributed to clinical patients who are displaying psychotic symptoms, as it was not present in those exhibiting affective symptoms (Kaustio et al., 2002)

#### 5.1.4.3 P300 and induced depressed moods

It is clear that the investigation into the effects of depressed mood on the P300 component is compromised by the diverse nature of the disorder. If the affective and cognitive aspects of depressive episodes could be isolated and induced in individuals, then a clearer picture of the influence of depression upon the P300 can be elucidated. By employing a mood induction procedure, a depressive mood can be created which will have affective and cognitive features as the dominant experience, therefore allowing the influence of depression upon the P300 to be investigated.

#### **5.1.5 The Resource Allocation Theory and the P300 Component**

Regardless of the model used to describe the origins of the P300 component, the amplitude of the P300 has been shown to be proportional to the attentional resources applied to the task being undertaken by the individual (Polich et al., 1995; Kramer & Strayer, 1988; Wickens, Kramer, Vanesse, & Donchin, 1983). Therefore, if the P300 is seen as representing an attention orienting system (Mulder, 1986; Kok, 1990), then the amplitude indexes the energy expenditure required to switch attentional processes towards novel activity within the environment (Hansenne et al., 2000). Alternatively, if the P300 is viewed as an indication of activation which accompanies the processing of new contextual information about a stimulus and its incorporation into the existing neuronal representation of the stimulus within working memory, it can be assumed that variations in amplitude correspond to the quality of information processing conducted (Polich et al., 2000).

Should the experience of depression deplete the available resources then, as predicted by the resource allocation model, there would be insufficient resources available to efficiently re-orient attentional processes or update neuronal representations of the stimulus. Consequently, the reduced energy capacity would be reflected in a reduction in P300 amplitude. Equally, if there are sufficient resources to control the orientation of attention or memory manipulation, then the amplitude of the P300 component would not be sensitive to the experience of a depressed mood.

#### **5.1.6 Experimental Hypothesis**

In terms of cognitive complexity, the processes thought to underlie the P300 component can be seen as representing low level information processing. Furthermore, the neuronal generators of the P300 component do not share a common neuroanatomical basis with those involved with the experience of emotion. Therefore it is assumed that there will be sufficient resources available to efficiently perform the oddball task whilst the participant is experiencing an induced depressed mood. It is hypothesised that an induced depressed mood will have no effect upon the amplitude of the P300 component recorded over the parietal region of interest during an oddball task.

As the neuroanatomical correlates of depression lie within the prefrontal cortex ((Dolan et al., 1993), electrophysiological manifestations of the induced mood would be likely to occur within the frontal region of interest. Davidson's model (Davidson, 1998a) implicates asymmetric frontal activation in an individual showing increased negative mood, with hypoactivity associated with the left hemisphere. It is hypothesised that an induced negative mood will lead to altered activity recorded over frontal regions. This will be measured by comparing ERP asymmetries in frontal areas.



## **5.2 Methods**

### **5.2.1 Participants**

Eight participants, 4 female and 4 male, with an average age of 24.7 years. All participants gave signed consent prior to taking part in the study and they were paid for their participation. All participants had achieved a score of 9 or less on the Beck Depression Inventory (BDI, (Beck, 1987). Two participants did not pass the screening procedure for the study as they scored more than 9 on the BDI. As this represents mild to moderate depression (Beck, 1987), it was deemed inappropriate to include them in a mood manipulation study. All participants who failed to pass the selection criteria fully debriefed and informed that as their BDI score indicated they were feeling mildly unhappy it would be inappropriate to manipulate their mood.

### **5.2.2 Measures and Induction Procedures**

Two measures were used to assess mood levels during this study, the Beck Depression Inventory (BDI; Beck, 1987) and the Positive Affect and Negative Affect Schedule scale (PANAS; Watson et al., 1988).

The BDI consists of 21 symptoms and attitudes, subdivided into four or five evaluative statements, which are designed to assess the severity of depression in adolescents and adults (Beck, 1987). It is widely accepted that the BDI is able to detect possible depression in normal populations (Steer et al., 1985), therefore it has been used to screen participants prior to undergoing the mood induction procedure.

The PANAS consists of 20 adjectives which are designed to produce measures of Positive Affect (PA) and Negative Affect (NA) (Watson et al., 1988). The scales have been shown to be a reliable, valid and efficient method of assessing PA and NA (Watson et al., 1988).

The Velten mood induction procedure was used to produce a neutral and depressed mood. The use of this procedure is described in detail in Chapter 4, and the statements and instructions used within the neutral and depressed treatments are presented in Appendix 2.

### **5.2.3 Stimuli, Design and Procedure**

A repeated measure design was employed, where participants performed an oddball task under a neutral and depressed condition. Performance of the neutral and depressed conditions was separated by at least one day, however the experimental procedure followed during each condition was identical. Upon completion of the depressed condition, participants took part in the elation treatment from the Velten mood induction procedure (see Appendix 2) in order to reverse any remaining depressed mood.

Within each session, each participant completed one experimental block containing 200 trials (40 targets, 160 standards). The presentation of stimulus type was randomised across the block.

Following the application of the Geodesic Sensor Net, participants completed a mood induction treatment and an oddball task. Performance of the neutral and depressed mood induction treatments was counterbalanced across the participants. During the oddball task, participants were instructed to press a key if they saw an 'X' and to make no response if

they saw a 'O'. A mood assessment was performed prior to induction, following induction and at the end of the oddball task.

Presentation of the mood induction statements and the oddball task stimuli (Target = X and standard = O) was controlled by the E-Prime experimental control system (Psychological Software Tools). Statements and stimuli were presented upon a RM PC-5200 Professional Multimedia computer, with a 15-inch screen.

#### **5.2.4 EEG/ERP Methods**

Scalp voltages were collected with a 128-channel Geodesic Sensor Net™ (Tucker, 1993), connected to a AC-coupled, 128-channel high input impedance amplifier (200M $\Omega$ , Net Amps™, Electrical Geodesics, Eugene, OR). Amplified analogue voltages (0.1Hz lowpass; 100 Hz highpass) were digitised at 250 Hz. Individual sensors were adjusted until impedances were less than 30 K $\Omega$ .

Trials were rejected from analysis if they contained more than 5 bad channels (changing more than 100  $\mu$ V between samples, or reaching amplitudes over 200  $\mu$ V). A moving window detection algorithm (NetStation™, Electrical Geodesics, Eugene, OR) marked channels containing vertical or horizontal eye movements (EOG channel differences greater than 70  $\mu$ V). A digital 30 Hz low pass filter was applied to the EEG recording prior to segmentation.

ERPs were computed within four different categories: neutral targets, neutral standards, depressed targets, depressed standards. Table 5.2 shows the number of trials included in the

ERP for each participant. It can be seen that data from one participant was rejected due to the presence of too few observations to form reliable ERPs.

Participant	Neutral target	Neutral standard	Depressed target	Depressed standard
*1	90	21	108	17
2	156	39	154	38
3	158	40	157	39
4	157	39	158	39
5	142	35	155	39
6	159	39	156	38
7	145	38	160	39
8	151	36	146	37

**Table 5.2** The number of trials included within the ERPs calculated for the neutral target, neutral standard, depressed target and depressed standard conditions for each participant.

An eye movement correction algorithm (Technical Appendix 2) was applied to trials contaminated with eye movements. All signal processing was conducted with a purpose-built signal processing toolbox for MatLab (MathWorks, Inc) (Technical Appendix 3). ERPs were baseline corrected with respect to a 100ms pre-stimulus recording interval. An average-rereference transformation was used to minimize the effects of reference site activity (Curran & Cleary, 2003).

Using a modification of the regions of interest defined by (Curran, Tucker, Kutas, & Posner, 1993b), ERPs were spatially average across the left frontal, left central, left parietal, left temporal, left occipital, right frontal, right central, right parietal, right temporal, and

right occipital regions of interest. Sensors included in each region are detailed in Technical Appendix 3.

### **5.2.5 Statistical Analysis**

Scores from the negative affect and positive affect sub-scales of the PANAS were analysed using a repeated measures analysis with mood induction procedure (neutral, depressed) and test time (pre-induction, post-induction, post task) included as factors.

The behavioural data were analysed using a repeated measures analysis, with mood (neutral, depressed) included as the factor. Reaction times for correct response to, and errors associated with target stimuli will be included in the analysis.

The peak amplitude of event-related waveform components were analysed using a repeated measures analysis, with mood (neutral, depressed), stimulus type (standard, target), hemisphere (left, right), region of interest (frontal, central, parietal, occipital) and time bin (250-350, 350-450) included as factors.

## **5.3 Results**

### **5.3.1 Mood Induction Procedure**

Both the negative affect and positive affect sub-scales were analysed using a repeated measures analysis with mood induction procedure (neutral, depressed) and test time (pre-induction, post-induction, post task) included as factors. Where significant effects were found, the relationship between levels was investigated using a Bonferroni corrected pairwise comparison.

5.3.1.1 Negative affect

The estimated groups means and standard errors for the negative affect scores during each mood induction procedure are presented in Table 5.3. The difference in negative affect during the two mood induction procedures was not significant ( $F(1,6) = 2.054, p = > 0.05$ ).

Mood induction	Mean (N = 7)	SE
Neutral	14.57	2.10
Depressed	15.57	1.83

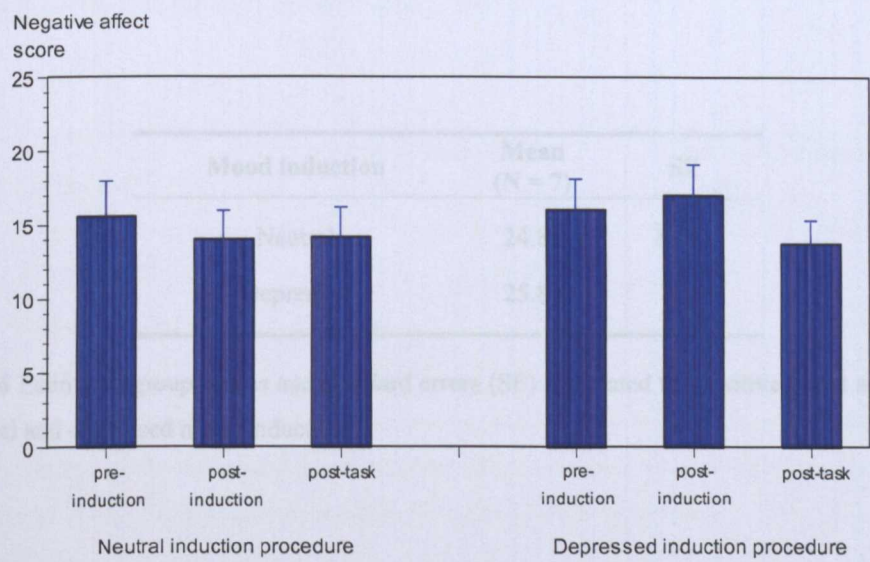
**Table 5.3** Estimated group means and standard errors (SE) calculated for negative affect scores under the neutral and depressed mood inductions.

The estimated groups means and standard errors for the negative affect scores at each time point are presented in Table 5.4. There was a significant main effect of test time ( $F(2,12) = 7.754, p = 0.007$ ), which reflected a decrease in negative affect as the experimental session continued. A pairwise comparison of the main effect revealed that the reduction in negative affect from post-induction to post-task was almost significant ( $p = 0.06$ ).

Time point	Mean (N = 7)	SE
Pre-induction	15.79	2.07
Post-induction	15.50	1.94
Post-task	13.93	1.72

**Table 5.4** Estimated group means and standard errors (SE) calculated for negative affect scores at the three time points during the experimental session.

There was a significant interaction between mood induction procedure and test time ( $F(2,12) = 3.709, p = 0.05$ ) (Figure 5.2). However this interaction does not elucidate anything meaningful in terms of the hypothesis under investigation. Estimated group means and standard errors for this interaction are presented in Table 5.5.



**Figure 5.2** Negative affect results from the mood assessments performed pre-induction, post-induction and nost-task for the neutral and depressed mood induction procedures.

Time point	Neutral Induction		Depressed induction	
	Mean (N = 7)	SE	Mean (N = 7)	SE
Pre-induction	15.57	2.34	16.00	2.01
Post-induction	14.00	1.92	17.00	2.04
Post-task	14.14	1.98	13.71	1.51

**Table 5.5** Estimated group means and standard errors (SE) calculated for negative affect scores at the three time points during the experimental session for each induction procedure.

5.3.1.2 Positive affect

The estimated groups means and standard errors for the positive affect scores during each mood induction procedure are presented in Table 5.6. The difference in positive affect during the two mood induction procedures was not significant ( $F(1,6) = 2.054, p = > 0.05$ ).

Mood induction	Mean (N = 7)	SE
Neutral	24.81	1.321
Depressed	25.81	2.40

**Table 5.6** Estimated group means and standard errors (SE) calculated for positive affect scores under the neutral and depressed mood inductions.

The estimated group means and standard errors for positive affect at each time point are presented in Table 5.7. There was a significant main effect of test time ( $F(2,12) = 6.282, p = 0.01$ ), which reflected a decrease in positive affect as the experimental session progressed. A pairwise comparison of this main effect did not reveal any significant differences.

Time point	Mean (N = 7)	SE
Pre-induction	28.429	0.869
Post-induction	25.429	1.965
Post-task	22.071	2.649

**Table 5.7** Estimated group means and standard errors (SE) calculated for positive affect scores at the three time points during the experimental session.



The interaction between mood and test time was not significant ( $F(2,12) = 0.829, p = > 0.05$ ), the estimated group means and standard errors for which are presented in Table 5.8. Under both mood induction procedures there is a reduction in the positive affect experienced by the participants across the experimental session. Additionally, carrying out the task produced a further decline in positive affect.

Time point	Neutral Induction		Depressed induction	
	Mean (N = 7)	SE	Mean (N = 7)	SE
Pre-induction	28.14	0.83	28.71	1.66
Post-induction	25.43	1.79	25.43	2.71
Post-task	20.86	2.12	23.29	3.52

**Table 5.8** Estimated group means and standard errors (SE) calculated for positive affect scores at the three time points during the experimental session for each induction procedure.

### 5.3.2 Behavioural Data

The behavioural data were analysed using a repeated measures analysis, with mood (neutral, depressed) included as the factor. Only correct responses to target stimuli were analysed, error rates were negligible and therefore omitted from the analysis.

The mean reaction times for correct responses to target stimuli under neutral mood and depressed mood conditions are presented in Table 5.9. There was no significant difference between the reactions times taken to respond to target stimuli under the neutral and depressed mood conditions ( $F(1,7) = 1.174, p = > 0.05$ ).

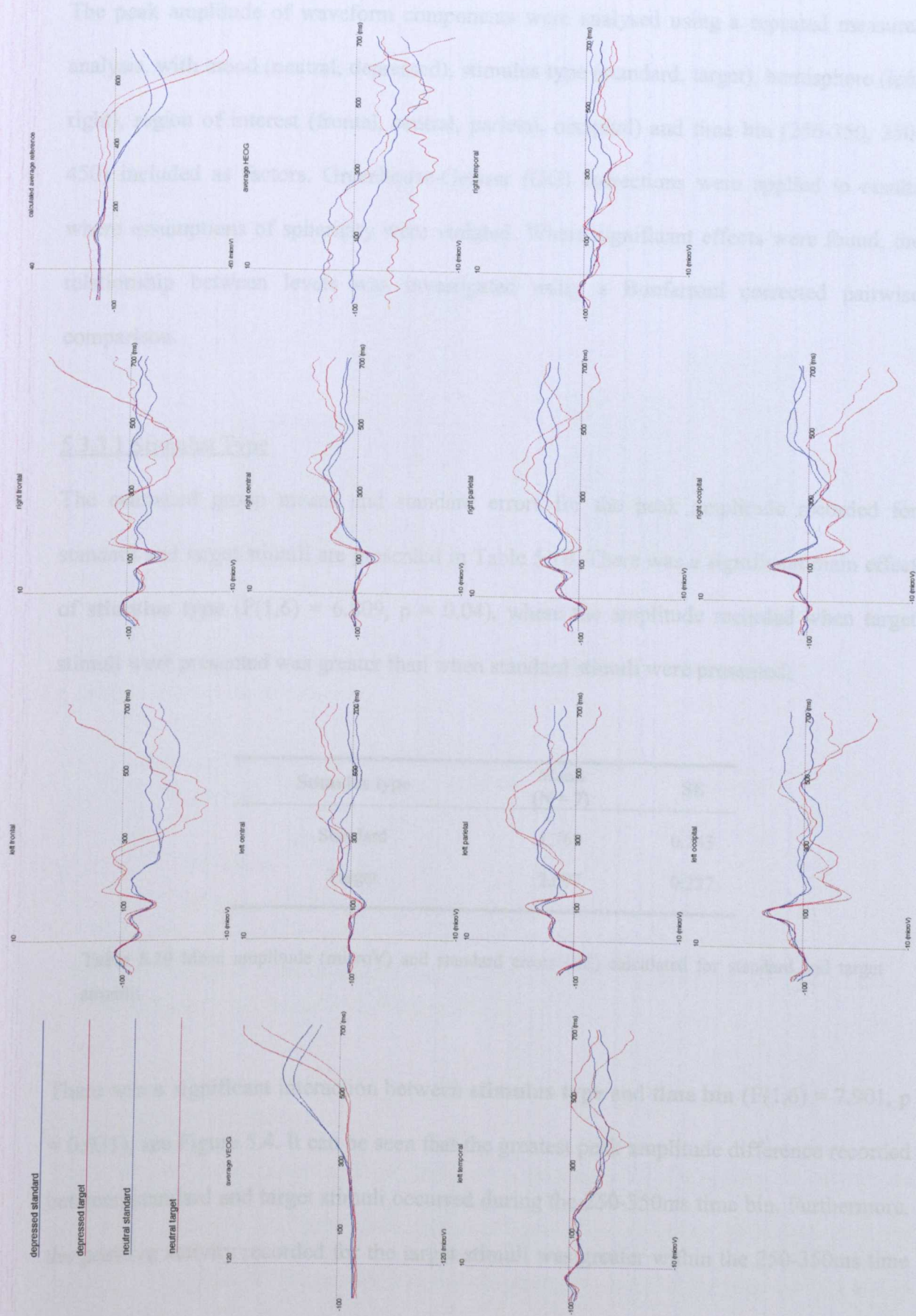
Mood induction	Mean (N = 8)	Standard Error
Neutral	373.68	16.54
Depressed	365.49	21.65

**Table 5.9** Mean reaction times (ms) and standard errors (SE) for target stimuli during the neutral mood and depressed mood conditions.

### 5.3.3 Electrophysiological data

Figure 5.3 shows the grand averaged event-related potentials for standard and target stimuli recorded under the neutral and depressed mood conditions. Each graph represents the activity recorded for 100 ms prior to stimulus presentation and 700 ms after stimulus presentation. As can be seen, there is a distinct difference between the standard and target waveforms over the frontal and parietal regions recorded under both the neutral and depressed conditions. The maximum deflections occur between 250ms and 450ms after stimulus presentation and have a negative polarity over the frontal regions and a positive polarity in more posterior regions.

The time of maximal activation was noted within the grand average ERPs and then checked within the individual average ERPs to ensure consistency across participants. Consequently, the peak amplitude of the maximum deflection within the 250-350ms and 350-450ms time bins was determined using an algorithm within the purpose-built signal processing toolbox (Technical Appendix 3).



**Figure 5.3** The grand averaged event-related potentials for standard and target stimuli recorded under the neutral and depressed mood conditions.

The peak amplitude of waveform components were analysed using a repeated measures analysis, with mood (neutral, depressed), stimulus type (standard, target), hemisphere (left, right), region of interest (frontal, central, parietal, occipital) and time bin (250-350, 350-450) included as factors. Greenhouse-Geisser (GG) corrections were applied to results where assumptions of sphericity were violated. Where significant effects were found, the relationship between levels was investigated using a Bonferroni corrected pairwise comparison.

### 5.3.3.1 Stimulus Type

The estimated group means and standard errors for the peak amplitude recorded for standard and target stimuli are presented in Table 5.10. There was a significant main effect of **stimulus type** ( $F(1,6) = 6.809$ ,  $p = 0.04$ ), where the amplitude recorded when target stimuli were presented was greater than when standard stimuli were presented.

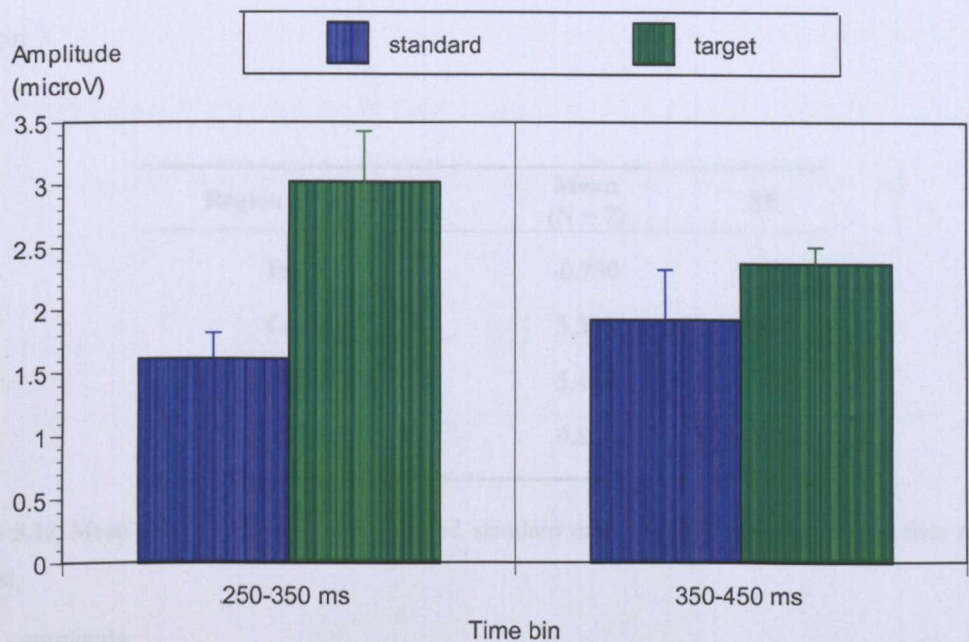
Stimulus type	Mean (N = 7)	SE
Standard	1.769	0.243
Target	2.697	0.227

**Table 5.10** Mean amplitude (microV) and standard errors (SE) calculated for standard and target stimuli.

There was a significant interaction between **stimulus type** and **time bin** ( $F(1,6) = 7.901$ ,  $p = 0.031$ ), see Figure 5.4. It can be seen that the greatest peak amplitude difference recorded between standard and target stimuli occurred during the 250-350ms time bin. Furthermore, the positive activity recorded for the target stimuli was greater within the 250-350ms time



bin than in the 350-450ms time bin. The group means and standard errors for this interaction are presented in Table 5.11.



**Figure 5.4** The mean peak amplitude (microV) recorded for standard and target stimuli within the 250-350ms and 350-540ms time bins.

Time bin	Standard		Target	
	Mean (N = 7)	SE	Mean (N = 7)	SE
250-350 ms	1.61	0.20	3.02	0.39
350- 450 ms	1.93	0.39	2.37	0.13

**Table 5.11** Mean peak amplitude (microV) and standard errors (SE) calculated for the standard and target stimuli within the 250-350ms and 350-450ms time bins.

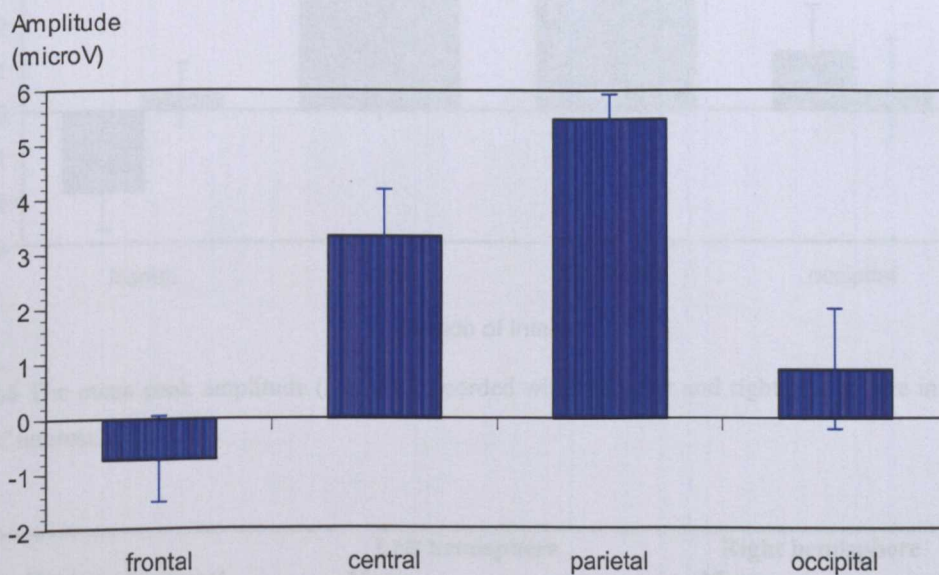
5.3.3.2 Regions of Interest

The estimated group means and standard errors calculated for the regions of interest are presented in Table 5.12. There was a significant main effect of **region of interest** ( $F(3,18) = 8.385, p = 0.009$  (GG corrected)) (Figure 5.5). It can be seen that the peak activity within

the regions of interest shifts in polarity across the scalp, the frontal region is associated with negative activation while the posterior regions are associated with positive activity. Furthermore, the parietal region of interest is associated with the greatest amplitude of activation.

Region of Interest	Mean (N = 7)	SE
Frontal	-0.730	0.778
Central	3.316	0.854
Parietal	5.466	0.458
Occipital	0.880	1.096

**Table 5.12** Mean peak amplitude (microV) and standard errors (SE) calculated for the four regions of interest.

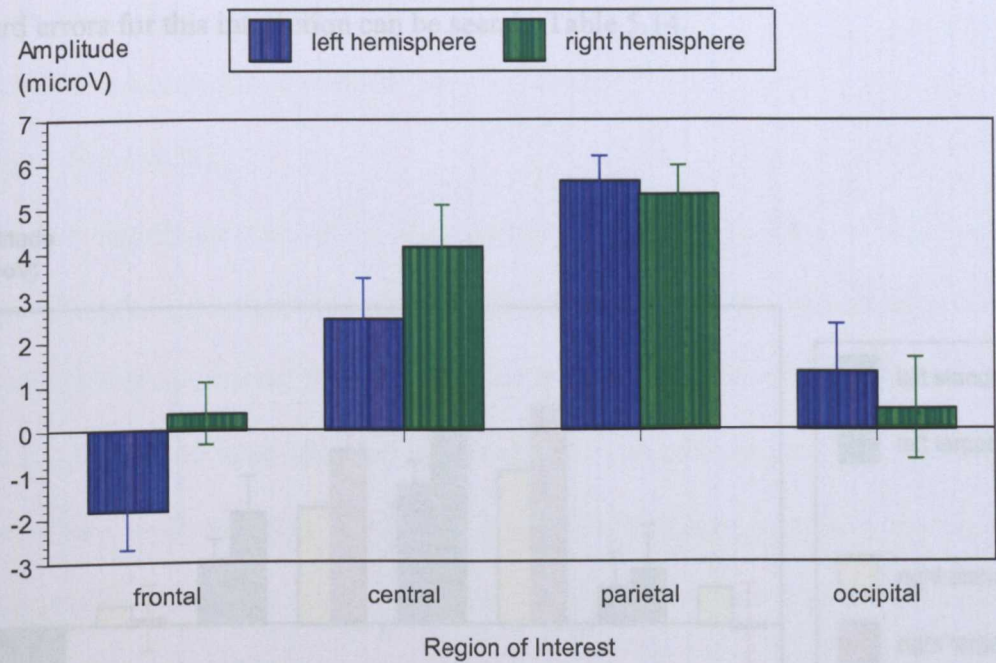


**Figure 5.5** The mean peak amplitude (microV) recorded over the four regions of interest.

There was a significant interaction between **region of interest** and **hemisphere** ( $F_{3,18} = 13.453$ ,  $p = < 0.001$  (GG corrected)) (Figure 5.6). It can be seen there are hemispheric



differences within all regions of interest, with the most marked difference evident in frontal areas. Within the posterior regions, although the strength of activation varies between the hemispheres and the regions of interest, the peak amplitudes reflect positive activity. However, in the frontal region of interest the left hemisphere is associated with negative activity while the right hemisphere is associated with little change in positive activity. The group means and standard errors for this interaction are presented in Table 5.13.

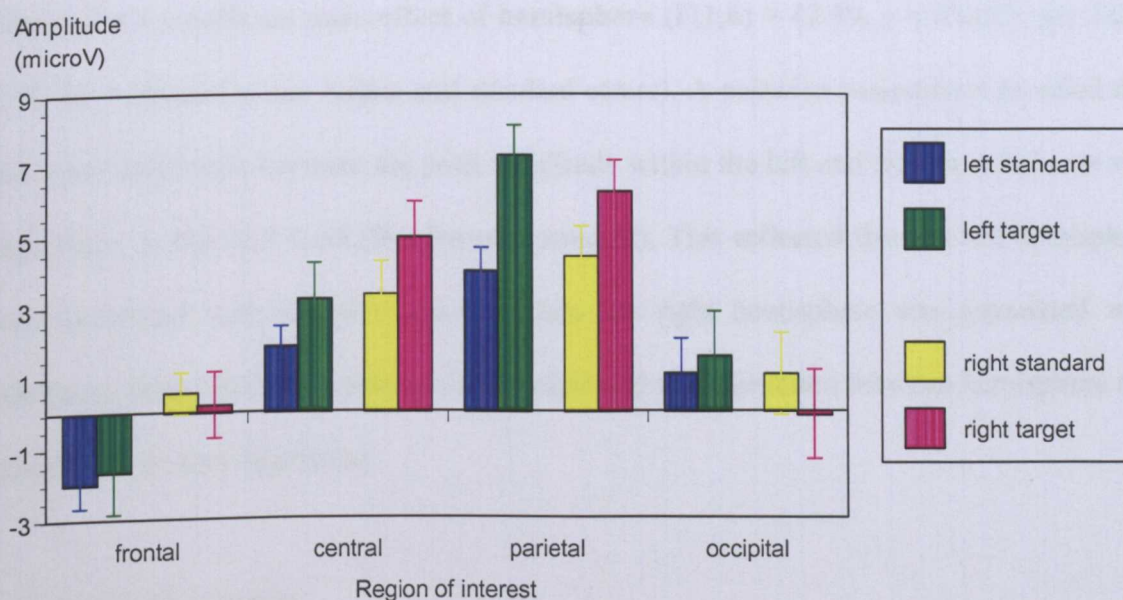


**Figure 5.6** The mean peak amplitude (microV) recorded within the left and right hemisphere in each of the regions of interest.

Region of interest	Left hemisphere		Right hemisphere	
	Mean (N = 7)	SE	Mean (N = 7)	SE
Frontal	-1.82	0.88	0.36	0.71
Central	2.50	0.91	4.13	0.94
Parietal	5.62	0.55	5.31	0.65
Occipital	1.30	1.06	0.46	1.14

**Table 5.13** Mean peak amplitude (microV) and standard errors (SE) calculated for the left and right hemispheres within the four regions of interest.

This region of interest and hemisphere interaction is further modified by **region of interest**, **hemisphere** and **stimulus type**, ( $F(3,18) = 4.329$ ,  $p = 0.05$  (GG corrected)) (Figure 5.7). Within the parietal region, the difference between peak positive amplitude for standard and target stimuli is greatest within the left hemisphere. Within the frontal region of interest activity recorded within the right hemisphere is positive and that recorded in the left hemisphere is negative. To explore these interactions further, a repeated measures ANOVA was performed upon the data within each region of interest. The estimated group means and standard errors for this interaction can be seen in Table 5.14



**Figure 5.7** The mean peak amplitude (microV) recorded for standard and target stimuli within the left and right hemispheres of the regions of interest.



		Frontal		Central		Parietal		Occipital	
		Mean (N = 7)	SE	Mean (N = 7)	SE	Mean (N = 7)	SE	Mean (N = 7)	SE
Standard	Left	-1.99	0.69	1.83	0.87	3.98	0.66	1.05	0.98
	Right	0.53	0.86	3.32	0.94	4.39	0.85	1.03	1.17
Target	Left	-1.66	1.19	3.17	1.03	7.27	0.85	1.55	1.27
	Right	0.19	0.94	4.94	1.01	6.23	0.89	-0.11	1.26

**Table 5.14** Mean peak amplitude (microV) and standard errors (SE) calculated for standard and target stimuli within the left and right hemispheres of the four regions of interest.

#### *5.3.3.2.1 ROI x hemisphere x stimulus post hoc analysis*

##### *5.3.3.2.1.1 Frontal ROI*

There was a significant main effect of **hemisphere** ( $F(1,6) = 42.89$ ,  $p = 0.001$ ) (see Table 5.12 for estimated group means and standard errors). A pairwise comparison revealed that the mean difference between the peak amplitude within the left and right hemispheres was significant at the 0.05 level (Bonferroni corrected). This reflected that the left hemisphere was associated with negative activity while the right hemisphere was associated with positive activity. Both the stimulus main effect and the interaction between hemisphere and stimulus were non significant.

##### *5.3.3.2.1.2 Central ROI*

There was a significant main effect of **stimulus type** ( $F(1,6) = 10.44$ ,  $p = 0.01$ ), the estimated group means and standard errors for which are presented in Table 5.15. A pairwise comparison revealed that the mean difference between peak amplitude recorded during standard and target stimuli was significant at the 0.05 level (Bonferroni corrected). This reflected that the amplitude of positive activity in response to target stimuli was

greater than that in response to standard stimuli. Both the hemisphere main effect and interaction between hemisphere and stimulus were non significant.

Stimulus type	Mean (N = 7)	SE
Standard	2.577	0.848
Target	4.055	0.919

**Table 5.15** Mean peak amplitude (microV) and standard errors (SE) calculated for standard and target stimuli within the central region of interest.

#### 5.3.3.2.1.3 Parietal ROI

There was a significant main effect of **stimulus type** ( $F(1,6) = 5.66$ ,  $p = 0.05$ ), the estimated group means and standard errors for which are presented in Table 5.16. A pairwise comparison revealed that the mean difference between peak amplitude recorded during standard and target stimuli was significant at the 0.05 level (Bonferroni corrected). This reflected that the amplitude of positive activity in response to target stimuli was greater than that in response to standard stimuli.

Stimulus type	Mean (N = 7)	SE
Standard	4.18	0.649
Target	6.747	0.760

**Table 5.16** Mean peak amplitude (microV) and standard errors (SE) calculated for standard and target stimuli within the parietal region of interest.

The main effect of hemisphere was non significant, however there was a significant interaction between **hemisphere** and **stimulus type** ( $F(1,6) = 8.138$ ),  $p = 0.02$ ) (see Table

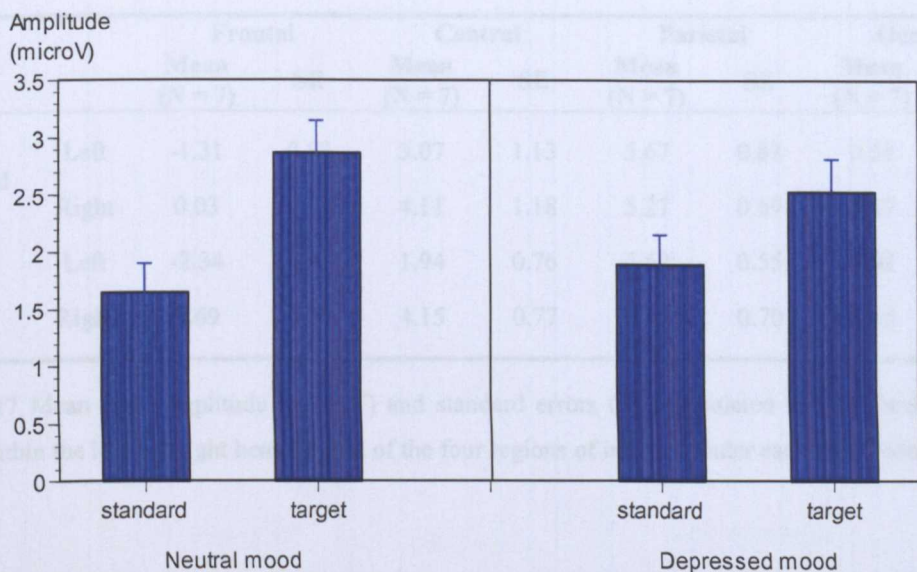
5.14 for the estimated group means and standard errors). It can be seen in Figure 5.7 that the peak amplitude difference between standard and target responses is greater within the left hemisphere than within the right.

#### *5.3.3.2.1.3 Occipital ROI*

There was a significant main effect of **hemisphere** ( $F(1,6) = 10.140$ ,  $p = 0.01$ ) (see Table 5.13 for estimated group means and standard errors). A pairwise comparison revealed that the mean difference between the peak amplitude within the left and right hemispheres was significant at the 0.05 level (Bonferroni corrected). This reflected that the peak positive amplitude within the left hemisphere was greater than that within the right hemisphere. Both the hemisphere main effect and interaction between hemisphere and stimulus were non significant.

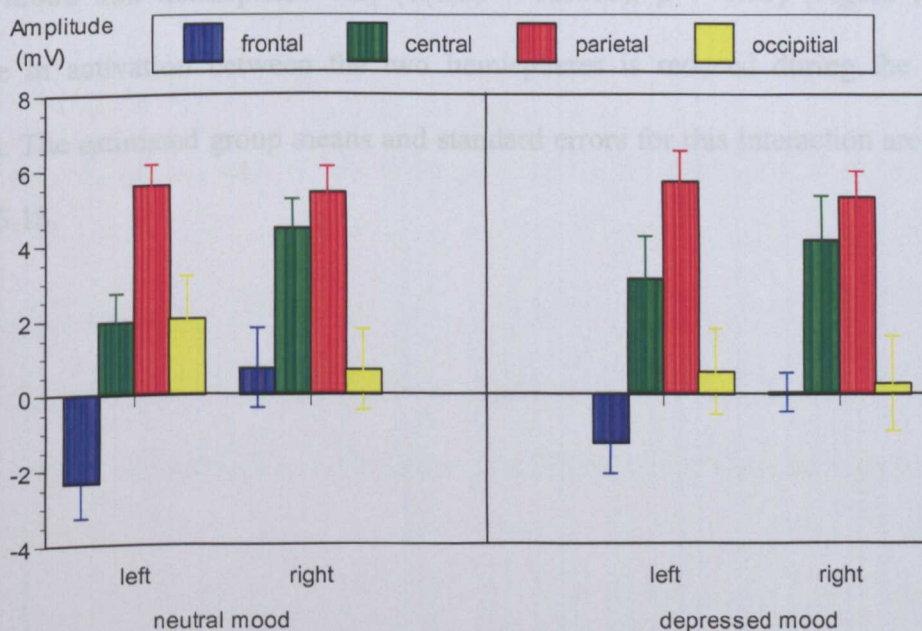
#### 5.3.3.3 Mood

Although not significant, it is worth pointing out the interaction between **mood** and **stimulus type** ( $F(1,6) = 9.571$ ,  $p = 0.06$ ) (Figure 5.9). It can be seen that the difference between the standard and target trials is reduced in the depressed mood condition, which is associated with a small increase in the amplitude of response to standard stimuli and a decrease in the amplitude of response to target stimuli.



**Figure 5.9** The mean peak amplitude (microV) associated with standard and target trials, recorded under neutral and depressed mood conditions.

There was a significant interaction between **mood**, **region of interest** and **hemisphere** ( $F(3,18) = 3.773, p = 0.05$  (GG corrected)) (Figure 5.10). To explore this further, a repeated measures ANOVA was performed upon the data within each region of interest. The group means and standard errors for this interaction can be seen in Table 5.17.



**Figure 5.10** The mean peak amplitude (microV) associated with left and right hemispheres within each of the region of interest, recorded under neutral and depressed mood conditions.

		Frontal		Central		Parietal		Occipital	
		Mean (N = 7)	SE	Mean (N = 7)	SE	Mean (N = 7)	SE	Mean (N = 7)	SE
Depressed	Left	-1.31	0.83	3.07	1.13	5.67	0.82	0.58	1.16
	Right	0.03	0.53	4.11	1.18	5.27	0.69	0.27	1.28
Neutral	Left	-2.34	0.96	1.94	0.76	5.59	0.55	2.02	1.14
	Right	0.69	1.07	4.15	0.77	5.36	0.70	0.65	1.09

**Table 5.17** Mean peak amplitude (microV) and standard errors (SE) calculated for standard and target stimuli within the left and right hemispheres of the four regions of interest, under each mood condition.

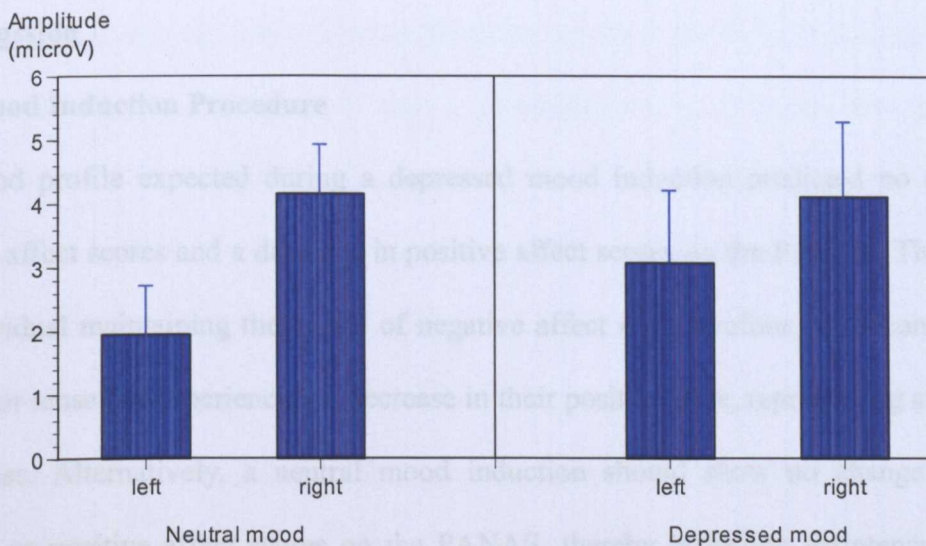
### 5.3.3.3.1 Mood x ROI x hemisphere post hoc analysis

#### 5.3.3.3.1.1 Frontal ROI

As previously reported, the main effect of **hemisphere** was significant ( $p = 0.001$ ). The mood main effect and interaction between mood and hemisphere were not significant.

#### 5.3.3.3.1.2 Central ROI

The main effects of mood and hemisphere were not significant, however the interaction between **mood** and **hemisphere** was ( $F(1,6) = 12.016$ ),  $p = 0.01$ ) (Figure 5.11). The difference in activation between the two hemispheres is reduced during the depressed condition. The estimated group means and standard errors for this interaction are presented in Table 5.18.



**Figure 5.11** The mean peak amplitude (microV) associated with left and right hemispheres within the central region of interest, recorded under neutral and depressed mood conditions.

Mood	Left hemisphere		Right hemisphere	
	Mean (N = 7)	SE	Mean (N = 7)	SE
Neutral	1.94	0.76	4.15	0.77
Depressed	3.07	1.133	4.11	1.17

**Table 5.18** Mean peak amplitude (microV) and standard errors (SE) calculated for activation within the left and right hemispheres of the central region of interest under neutral and depressed moods.

#### 5.3.3.3.1.3 Parietal ROI

There were no significant main effects or interactions within this region of interest.

#### 5.3.3.3.1.4 Occipital ROI

As previously reported, the main effect of **hemisphere** was significant ( $p = 0.01$ ). The mood main effect and interaction between mood and hemisphere were not significant.



## **5.4 Discussion**

### **5.4.1 Mood Induction Procedure**

The mood profile expected during a depressed mood induction predicted no change in negative affect scores and a decrease in positive affect scores on the PANAS. This reflects the individual maintaining their state of negative affect and therefore not becoming more anxious or tense but experiencing a decrease in their positive state, representing an increase in sadness. Alternatively, a neutral mood induction should show no change in either negative or positive affect scores on the PANAS, thereby reflecting maintenance of the individual's baseline mood state.

Although there were no significant changes in the PANAS scores, there were trends in the predicted direction, with the neutral mood induction procedure associated with a small decrease in both negative and positive affect, while the depressed procedure associated with an increase in negative affect and a decrease in positive affect. The small changes in mood registered by the negative and positive affect sub-scales of the PANAS did not correspond to the participants' reports upon completion of the experimental sessions.

Prior to leaving the laboratory, participants were asked to describe their mood during the session. All reported that they felt sadness during the depressed condition. However, the changes recorded during the depressed condition appear to underestimate the strength of their emotions. Alternatively, participants reported that they found the neutral mood induction procedure somewhat boring. The small decreases in negative and positive affect associated with the neutral condition could reflect the participants' feelings of boredom.

One possibility is that the PANAS is insufficiently sensitive to the mild or transient mood changes manipulated here. There is also a possibility that participants rationalise their emotions when they come to complete the mood assessment. Although they are experiencing a sadness induced by the procedure, they are aware that it is a transient experience and they will soon be feeling 'normal' again. Although participants are instructed to indicate their mood 'at this point in time', it is possible they adjust their perception of their mood to account for their knowledge that their original mood state will soon be re-experienced.

Therefore, although there is a slight difference in mood profiles associated with the two mood induction procedures, the PANAS scores do not indicate distinct differences between neutral and depressed mood.

#### **5.4.2 Behavioural Data**

There was no difference between the two 'mood' conditions with respect to performance on the oddball task. Within clinical populations, there is a slowing of reaction time associated with depressed individuals, but it is not surprising that the mild altered mood state achieved here did not interfere with motor responses. Also, it is possible that the reduction in response times associated with depressed individuals is a consequence of chronic motivational and biological factors. Therefore a transient alteration in mood may not be sufficient to influence psychomotor control.

In addition, the Resource Allocation Model (Ellis et al., 1988) predicts that depression will be associated with performance deficits only when the experience of the emotions leads to there being insufficient resources available to perform cognitive task. As the processes



underlying the oddball task are associated with low levels of complexity, it had been postulated that there would be sufficient resources available to carry out this simple stimulus identification process.

#### **5.4.3 Electrophysiological Data.**

Before the influence of depressed mood can be assessed, it must be determined whether a traditional P300 component was elicited by the oddball paradigm used in this study. The morphology of this electrophysiological response reflects a difference in activity between the waveform associated with standard and target trials. This difference peaks at around 300 ms, with the waveform component associated with the target stimuli exhibiting a greater positivity than that associated with the standard stimuli. Furthermore, this peak difference is greatest in recordings made over the parietal regions of the scalp (Polich, 1999). It can be seen in these data that the grand averaged waveforms produced for this study (Figure 5.3), do show a clear P300 distinction over the left and right parietal regions of interest.

The statistical analysis of peak activity within these areas is consistent with the presence of a P300 waveform component. For example, the peak activity recorded in response to target stimuli was significantly more positive than the peak activity associated with standard stimuli ( $p = 0.04$ ), and the greatest difference in peak activity occurred within the 250-350 ms time bin ( $p = 0.03$ ). Furthermore there was a significant main effect of region of interest ( $p = 0.009$ ), which indicated that the greatest levels of positive activity were associated with the parietal regions of interest.

It was hypothesised that, as the P300 is maximal over central and parietal regions, any cortical effects of induced depressed mood would not be reflected in this component. The neuronal generators of the P300 component do not share a common neuroanatomical basis with those frontal areas hypothesised to underpin the interaction between cognition and affect. As hypothesised, there were no differences in the P300 component measured in the two mood conditions, although, of course, this must be assessed in the light of the apparent failure of the mood induction procedure.

It is worth noting that, although not significant, there was an overall interaction in ERP amplitudes between mood and stimulus type ( $p = 0.06$ ). The amplitude difference in response to target and standard trials seen in the neutral condition was *reduced* under the depressed mood. The differentiation between the two stimuli types was associated with an increase in positive amplitude associated with the standards stimuli and a decrease in the positive amplitude associated with the target stimuli. A very speculative interpretation of this could be that this underpins reported failures to differentiate between significant and nonsignificant events characteristic of depressive mood (Davidson, Pizzagalli, Nitschke, & Putnam, 2002).

The secondary hypothesis predicted asymmetric changes in frontal activity as a function of mood (Dolan et al., 1993). There was a main effect of hemisphere within the frontal regions of interest ( $p = 0.001$ ). The left hemisphere was associated with negative activation, while the right hemisphere was associated with positive activation. Unfortunately, there was no difference in this overall asymmetry as a function of mood manipulation, although, of course, this manipulation was subjectively not successful as measured by the PANAS data.

There was a significant interaction between mood, region of interest and hemisphere ( $p = 0.05$ ), but a post hoc analysis of this interaction showed that it was the central region of interest that was sensitive to the modulation of mood. A lateralisation of activation seen in the neutral mood, where a greater positivity was associated with the right hemisphere, was reduced in the depressed condition. It can be seen in Figure 5.10 that the asymmetry is reduced by an increase in left hemisphere activation.

Overall, these findings point to there being a potential blurring of the stimulus and hemispheric differences associated with task performance under a depressed mood. As reported earlier, there were no differences with the reaction times associated with this particular task. However, it is possible that the maintenance of behavioural performance is through the compensatory recruitment of additional neuronal populations. The reduction in asymmetry reported here was associated with an *increase* in activation, on the left hemisphere. Even with the lack of subjective differences in mood state associated with this study, there is evidence that the neurological signature is different under the two mood states.

In summary, this initial study indicates some potential for investigating changes in cortical activation patterns associated with transient changes in mood, as, even with this very low level 'passive attention' task there is some evidence of differences between the two mood conditions which were not evident at the subjective or the behavioural level. The Resource Allocation model predicts that any changes there are will become more marked with increasing cognitive demands. The next study will include a task that is a) more cognitively demanding and b) more associated with activity in the frontal areas where cognition/affect interactions may be modulated.

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## **Chapter 6**

### **Mood and cognition II – The influence of depressed mood on a selective attention task**

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#### **6.1 Introduction**

Selective attention tasks can be described as having a moderate level of cognitive intensity. During successful performance an automatic response must be superseded, by way of selective attention, to allow the initiation of a controlled response. Therefore in addition to stimulus identification, information processing will require the representation and maintenance of the information necessary to adaptively control behaviour, a procedure referred to as context processing (MacDonald & Carter, 2003).

Neuroanatomical activation during tasks requiring context processing has been associated with the dorsolateral prefrontal cortex (George et al., 1994). As this cortical region is implicated in the experience of depression, and the tasks have a moderate level of cognitive intensity it is possible that selective attention tasks will be susceptible to the detrimental effects of depressed mood. However, additional processing of incongruent stimuli has been localised to the semantic processing within the temporo-parietal region (Liotti et al, 2000). Therefore the distribution of cortical activation may allow the maintenance of efficient performance during a depressed mood. To allow the

electrophysiological investigation of this hypothesis, the Stroop task (Stroop, 1935) will be performed under neutral and depressed moods.

### **6.1.1 The Stroop Effect**

The Stroop task is one of the most widely studied examples of a selective attention task (Siegle, Steinhauer, & Thase, 2004). In the classic paradigm, participants are instructed to name the ink colour that colour words are presented in. Its elegance lies in the fact that simplicity of the task conceals how strongly the colour word interferes with the act of identifying the ink colour. Consequently it produces one of the most robust effects in psychology (Ilan & Polich, 1999). Shorter response times are recorded for words where the ink colour and meaning are congruent (e.g. the word 'blue' in blue ink), producing the Stroop facilitation effect. While longer response times are associated with words with incongruent ink colour and meaning (e.g. the word 'blue' in yellow ink), resulting in the Stroop interference effect (Stroop, 1935).

Although considered a Stroop phenomenon, the presence of facilitation is not necessarily concurrent with interference, and if present, its existence is usually insubstantial in comparison to the interference effect (MacLeod, 1991). The inconsistency of the facilitation effect is thought to reflect the difficulty in accelerating an inherently swift response (MacLeod, 1991; Glaser & Glaser, 1982). Consequently the many theories proposed to account for the Stroop phenomenon, and this thesis, will concentrate upon the interference effect.

### **6.1.2 Theories of Stroop Interference Effect**

Explanations of the Stroop effect can be categorised in terms of where in the information processing sequence the interference occurs. The two possible locations are therefore, during early categorisation of the stimulus or during response generation.

#### **6.1.2.1 Stimulus Categorisation**

Theories of early processing interference are epitomised by the perceptual encoding hypothesis (Hock & Egeth, 1970), which proposes that the automatic processing of the word meaning distracts attention away from the ink colour. The majority of theories however, allude to the formation of the correct response as being the loci of the interference (Morton, 1969; Keele, 1972; Warren, 1972; Posner & Snyder, 1975).

#### **6.1.2.2 Response Generation**

A common assumption within these response competition hypotheses is that all salient dimensions of the stimulus are processed in parallel, and each is associated with a motor program controlling the appropriate response. During congruent trials, both the word and ink colour are associated with the same motor response so there is no cognitive cost involved. However, incongruent trials are associated with conflicting motor response programs, therefore the interference effect reflects the resolution of the response competition.

#### *6.1.2.2.1 Speed of Processing*

Early theories proposed that the relative speed of processing was responsible for the increase in reaction time associated with the interference effect (Morton & Chambers, 1973). Although stimulus feature processing occurs in parallel, it does so at different speeds (Atkinson, Drysdale, & Fulham, 2003). As the response program can only be initiated by one channel of information, there is an inherent tendency for this to be done by the word meaning as this dimension is processed more efficiently than ink colour.

#### *6.1.2.2.2 Strength of Processing*

More recently, the parallel distributed processing model (Cohen, Dunbar, & McClelland, 1990) has become the dominant explanation of the interference effect. This model places the emphasis upon the strength of activation during the parallel processing of word meaning and ink colour, rather than the temporal dynamics (Atkinson et al., 2003). As the initiation of a response will be dependent upon the intensity of the competing input channels (MacLeod, 1991), increased attention due to the automaticity of reading the word leads to strongest pattern of activation being associated with this stimulus dimension (Atkinson et al., 2003).

Therefore, the correct performance of the Stroop task requires the participant to actively ignore the irrelevant stimulus dimension (word meaning) and its associated response. Overriding this attentional bias implicates the activation of a selective attention system. Although it is widely accepted that an anterior attentional system plays an integral role in the Stroop task (Zysset, Müller, Lohmann, & Yves von Cramon, 2001; Milham et al.,

2001), the identification of the cortical regions which subserve such a system is a more contentious issue.

### **6.1.3 The Neuroanatomical Basis of the Stroop effect**

#### **6.1.3.1 The Anterior Cingulate Cortex**

One area thought to be associated with the task is the anterior cingulate cortex (ACC; Zysset et al., 2001). In particular, task specific activation has been recorded in the cognitive subsection of this structure (Pardo, Pardo, Janer, & Raichle, 1990; Bench et al., 1993; George, Ketter, Parekh, Rosinsky, & Ring, 1994; Carter, Mintum, & Cohen, 1995; Derbyshire, Vogt, & Jones, 1998; Gruber, Rogowska, Holcomb, Soraci, & Yurgelun-Todd, 2002). This functional association was seen to be strengthen further by the activation of the ACC in qualitatively similar tasks, such as divided attentions tasks (Corbetta, Miezin, Dobmeyer, Shulman, & Petersen, 1991) and response selection/generation tasks (Kawashima et al., 1996; Paus, Petrides, Evans, & Meyer, 1993). Consequently, it was hypothesised that this consistent activation of the ACC was evidence of its integral role in the resolution of response conflicts (Zysset et al., 2001).

#### **6.1.3.2 The Prefrontal Cortex**

Recently, the focus has shifted to the involvement of both the ACC and the PFC in the Stroop effect (Banich et al., 2000b; Banich et al., 2000a; Gehring & Knight, 2000; MacDonald, Cohen, Stenger, & Carter, 2000). This alternative explanation suggests a more supportive role for the anterior cingulate, acting as a motor response co-ordinator for lateral prefrontal cortex mediated judgements (Paus et al., 1993; Paus, Koski, Caramanos, & Westbury, 1998).



Furthermore, evidence of a somatotopically organised ACC suggests that each output modality may be represented by a different region (Paus et al., 1993; Picard & Strick, 1996). Initial studies supporting this view were centred upon the preparation and execution of motor activity within non-human primates (Paus et al., 1993; Shima et al., 1991), however there is growing evidence of a similar functional organisation within humans. For example, focal lesions to the mid/caudal regions of the ACC do not interfere with performance of the Stroop task when a verbal response is required, but prevents the transposition into a manual response (Turken & Swick, 1999).

Although aiming to validate a primary functional association between the ACC and the Stroop effect, Zysset et al (2001) ultimately provided support for the secondary role described above. Using a modified Stroop task, participants were instructed to indicate whether the ink colour of the word presented in trial 1 matched the meaning of the word presented in trial 2. As a result the conceptual interference is separated from the preparation of the motor response, and therefore the response conflict (Zysset et al., 2001). Following from the proposition that the ACC is responsible for the facilitation and suppression of motor responses during such a conflict (Turken & Swick, 1999), it is not surprising that no activation of the ACC was reported by Zysset et al, (2001).

Zysset et al (2001) reported that the cortical region associated with the strongest activation was the lateral prefrontal cortex. The association between the lateral PFC and the interference effect was strengthened further when it was shown to be the only region to remain activated when congruent and incongruent conditions were compared (Zysset et al., 2001).

Consequently, Zysset et al (2001) argued that the lateral prefrontal cortex is responsible for 'task set management', a role which includes regulating response selection and ensuring that attention is focused upon the appropriate stimulus dimension. Citing support from neuroimaging studies investigating task switching (Dove, Pollmann, Schurbert, Wiggins, & von Cramon, 2000), cognitive flexibility (Konishi et al., 1998), and go/no-go tasks (Konishi, Nakajima, Uchida, Sekihara, & Miyashita, 1998), Zysset et al (2001) concur with the view that the inferior frontal cortex is responsible for information selection in situations where conflicting options are available (Thompson-Schill, D'Eposito, Aguirre, & Farah, 1997).

#### 6.1.3.3 A Duel Processing Model of Stroop Interference

Further evidence of the PFC and ACC being differentially involved in attentional control during the Stroop task was provided by Milham et al (2001). In this study, incongruent trials were manipulated so that the colour words were either presented in ink colours associated with a possible response (an eligible response) or colours outside of the possible responses series (an ineligible response) (Milham et al., 2001). Therefore during eligible response trials both conceptual and motor response interference could occur, while ineligible response trials would be associated with conceptual interference only. Activation of the ACC was shown to be sensitive to the type of interference, only being associated with eligible response trials (Milham et al., 2001). On the other hand, conflict within both the conceptual and motor response domains was associated with activation of the PFC (Milham et al., 2001).

The fine temporal resolution of electromagnetic neuroimaging techniques has provided another means of assessing the neurological signature of the Stroop interference effect.

Using magnetoencephalography (MEG) to determine patterns of event related desynchronisation (ERD) across the cortex, activation of the left dorsolateral PFC was seen to play a significant role in the processing of information during the Stroop task (Ukai et al., 2002). It should be noted that this study failed to show activation of the ACC, however this is likely to be attributed to the neuroanatomical location of the ACC causing weak magnetic signals rather than reflecting functional insignificance (Ukai et al., 2002).

Therefore, current neuroimaging studies suggest a joint role for the ACC and PFC in the Stroop task. Task management is thought to be mediated by the PFC (Zysset et al., 2001), with one of the main functions being to distinguish between task-relevant and task-irrelevant information (Milham et al., 2001). This attention system is then subserved by the ACC, which selectively regulates conflict within the response domain therefore its main aim is to prevent incorrect motor responses (Milham et al., 2001).

#### **6.1.4 Limitations of Haemodynamic Neuroimaging Techniques in Stroop Research**

Although PET and fMRI techniques have offered us much of our knowledge on the neuroanatomical substrates of the Stroop effect, their reliance upon block design paradigms limits their usefulness (Liotti, Woldorff, Perez, & Mayberg, 2000). The constraints enforced by this methodology result in blocks of congruent trials being compared with blocks of incongruent trials (for example: Derbyshire et al., 1998; McKeown et al., 1998). Liotti et al (2000) rightly argue that this creates quite a different task to the traditional Stroop design, where incongruent and congruent trials are randomly interspersed. Not only are the switching and unpredictability components removed, but the incongruent blocks are likely to rely upon a qualitatively different level of attention and information processing (Liotti et al., 2000).

One of the main advantages of electrophysiological research is that because segmentation and averaging occur off-line, different trial types can be randomly presented and then separated during the signal processing procedure. Therefore the classic Stroop paradigm can be used, allowing the neurological activity associated with frontal lobe controlled characteristics of the task to be measured and the true cognitive workload of the task to be assessed.

### **6.1.5 Electrophysiological investigations of the Stroop interference effect**

The real-time properties of electrophysiological recordings make them an ideal method of locating where in the signal processing sequence the Stroop interference effect occurs. As the morphology of ERP waveforms lend themselves to the differentiation between perceptual and cognitive processes (Rugg & Coles, 1996), the stimulus evaluation and response related processes integral to the theoretical explanation of interference can be disassociated (Donchin & Coles, 1988; Duncan-Johnson & Donchin, 1982). To this end, electrophysiological studies suggest that the period between ~300ms and 600ms post stimulus accommodates the modulation of brain activity caused by the interference effect (Ilan & Polich, 1999; West & Alain, 1999; Rebai, Bernard, & Lannou, 1997; Grapperon, Vidal, & Leni, 1998).

#### **6.1.5.1 Response Generation**

Event-related potential studies support the dominant theoretical viewpoint that Stroop interference occurs at the response output stage of information processing (Atkinson et al., 2003). As a result of its sensitivity to perceptual processes (Donchin, 1981), the P300 component is used to index the neurological activation surrounding stimulus categorisation (Ilan & Polich, 1999). However no differences in the early stages of the ERP have been

recorded (Atkinson et al., 2003; Ilan & Polich, 1999; West & Alain, 1999; Khateb, Michel, Pegna, Landis, & Annoni, 2000), indicating that the interference occurs at a cognitive rather than perceptual level (Ilan & Polich, 1999).

In addition, the absence of a latency effect for the P300 component implies that the time taken for stimulus evaluation under the congruent and incongruent conditions was comparable (Duncan-Johnson & Kopell, 1981; Atkinson et al., 2003). As the parallel processing of the two salient dimensions did not differ temporally, Atkinson et al (2003) concluded that the strength of neuronal activation, as posited in the parallel distributed processing theory (Cohen et al., 1990), underlies the Stroop effect.

#### 6.1.5.2 Strength of Processing

This consistency across studies has resulted in the main focus of electrophysiological investigations being placed upon activity within the later stages of the ERP waveforms. West & Alain (1999) concluded that there were four modulations which differentiated between incongruent and congruent trials:

- a phasic modulation which peaked around 500ms, positive over lateral fronto-polar regions and inverted over fronto-central regions;
- a slow wave beginning at about 500ms and persisting for duration of trial, manifest as negativity over fronto-central regions and positivity over fronto-polar regions;
- a decreased in positivity which peaked at 522ms over parietal regions;
- and finally a greater positivity peaking at around 650ms, over temporo-parietal regions (West & Alain, 1999).

This study allowed West & Alain (1999) to build up quite a complex picture of the neurological signature of the Stroop effect. The behavioural data indicated that the average response on incongruent trials was made at around 942ms, as opposed to 681ms for congruent trials (West & Alain, 1999). The first stage in the response process occurs at around 500ms, where a phasic modulation was recorded within anterior regions. The distribution of this activation across the scalp suggested the source generator was situated within the lateral prefrontal cortex, therefore supporting the involvement of this area in the Stroop task (West & Alain, 1999).

The continuation of the second modulation until the manual response was thought to reflect a conflict resolution process, associated with a deep neuronal generator such as the ACC (West & Alain, 1999). This supports hemodynamic neuroimaging studies which propose that the ACC is responsible for ensuring the correct motor response is initiated (Milham et al., 2001). The third modulation between 400 and 600ms over the parietal regions was virtually identical for the congruent and word identification trials, but was attenuated upon the incongruent trials. As congruent and word identification trials are thought to be associated with similar conceptual representations of word meaning (Kanne, Balota, Spieler, & Faust, 1998), the attenuated signal during the incongruent trial implies that this system is not utilised to the same degree (West & Alain, 1999). Alternatively, the attenuation reflects the suppression of the system when the conflicting conceptual representations for the ink colour and word meaning are activated (Lindsay & Jacoby, 1994).

Finally, the temporo-parietal late positive complex is thought to reflect the activity of a neural system which is involved in the processing of low level colour information (West &

Alain, 1999). Interestingly, the modulation becomes most pronounced during the time period between the average response times for the congruent and incongruent trials. West and Alain (1999) felt this reflected the detection of the incompatibility between the ink colour and the word meaning and therefore the point at which it becomes apparent that the conceptual system is unable to guide the response.

#### 6.1.5.2 Duel Processing Model of Stroop Interference

There are two important conclusions to be drawn from the work of West & Alain (1999). First is the existence of two frontally distributed modulations indicates there are two distinct processes associated with the Stroop effect that are governed by the lateral PFC and the ACC (West & Alain, 1999). Therefore adding further support to the fMRI and PET studies that suggest both these regions are associated with the Stroop task (Zysset et al., 2001; Milham et al., 2001).

Secondly, their findings support a model where word and colour pathways converge upon a single conceptual system which supports task performance (West & Alain, 1999). The Stroop interference effect reflects the suppression of the word pathway innervation upon detection of incompatibility, and the subsequent time required to achieve a sufficient level of activation within the colour pathway so that the correct response can be initiated (West & Alain, 1999). Again, providing support for the parallel distributed processing theory of the Stroop effect (Cohen et al., 1990).

Liotti et al (2000) provided further support for two temporally and spatially distinct processes underlying the Stroop interference effect. Using a 64 channel dense array acquisition system, an initial negativity within the 350-500ms post stimulus time period

was localised to a discrete anterior source generator (Liotti et al., 2000). The dipole source analysis suggested that there were two independent generators within the anterior cingulate cortex that were response modality specific, where verbal responses were associated with an anterior-medial distribution and manual response were associated with a medial dorsal distribution (Liotti et al., 2000). Therefore providing support for the postulation that the anterior cingulate cortex is somatotopically organised by response output modality (Paus et al., 1993; Picard & Strick, 1996).

The second component was a lateralised late positive complex which extended over the left temporo-parietal region within the 500-800ms post-stimulus time bin (Liotti et al., 2000). As this activation was present in all modality conditions, it was proposed to represent additional semantic processing of the incongruent stimuli (Liotti et al., 2000).

In contrast, Khateb et al (2000) reported that the only differentiation between incongruent and congruent trials occurred within the 300-400ms post-stimulus time period. The prolonged positive activation within right anterior regions was proposed to reflect the activation of the anterior cingulate cortex during the resolution of the interference (Khateb et al., 2000). It was concluded that the Stroop effect reflected increased demands placed upon attentional processes localised within the right anterior region, rather than perceptual or semantic processing (Khateb et al., 2000).

However, this study can be seen as supporting the role of anterior cingulate as a subordinate system which regulates conflict response (Milham et al., 2001). Although it is slightly earlier within the waveform, this activation is comparable to the positivity recorded over



fronto-polar regions by West & Alain (1999), therefore providing support for the dual process explanation of the Stroop interference effect.

#### **6.1.6 The Resource Allocation Model and Selective Attention**

Although a number of studies have found that depression disrupts performance of the Stroop task, the manifestation of impairment varies (Siegle et al., 2004; Ottowitz, Dougherty, & Savage, 2002). There is evidence of there being both increases in error rates (Lockwood, Alexopoulos, & van Gorp, 2002; Schatzberg et al., 2000) and reaction times (Degl'Innocenti, Agren, & Backmann, 1998; Lemelin, Baruch, Vincent, Evertett, & Vincent, 1997; Siegle et al., 2004) when depressed patients are compared to healthy controls.

Interestingly, Siegle et al (2004) utilised pupillary responses as an index of the degree of cognitive workload associated with the performance of the Stroop task by a depressed patient population. Pupil dilation has been reliably demonstrated as a physiological indicator of cognitive load (Siegle et al., 2004), with increases in attentional demand, memory processing and task difficulty all being associated with increases in pupil size (Beatty, 1982; Steinhauer & Hakerem, 1992). With particular reference to this thesis, it is thought that this pupillary response reflects the temporal dynamics of brain activity within regions associated with executive function, such as the dorsolateral prefrontal cortex (Siegle, Steinhauer, Stenger, Konecky, & Carter, 2003).

Although depressed participants did not show a pupillary response that was specific to Stroop interference, there was a decrease in pupil dilation in the seconds following stimulus presentation (Siegle et al., 2004). The application of a computational model suggested that

this pattern of response was consistent with a decrement in cognitive control caused by disruption to the functionality of the prefrontal cortex (Siegle et al., 2004). The model specifically postulated that insufficient innervation from the prefrontal cortex would lead to a decrement in task engagement, which could consequently lead to selective attention deficits (Siegle et al., 2004). Siegle et al (2004) concluded that the model's predictions were consistent with both the evidence of depressed individuals showing performance deficits on tasks driven by the prefrontal cortex (Ottowitz et al., 2002) and hypoactivity within the dorsolateral prefrontal cortex of depressed individual (for example, Bench et al., 1992; Drevets, 2001).

The Resource Allocation Model provides a possible explanation for this phenomenon. It is conceivable that the hypofrontality exhibited by the depressed individuals creates a smaller reserve of resources that can be applied to the wide range of tasks controlled by the dorsolateral prefrontal cortex. When engaged in a moderately intensive selective attention task, such as the Stroop task, there would be insufficient resources available to the control of the pupillary response. Therefore upon presentation of the stimulus there would be a momentary lapse in pupillary control whilst the available resources were utilised in the information processing associated with the task.

#### **6.1.7 Experimental hypothesis**

Given the involvement of frontal regions in Stroop-associated selective attention (Liotti, Woldorff, Perez, & Mayberg, 2000), it is assumed that behavioural and cortical indices of Stroop performance will be sensitive to mood changes. It is hypothesised that the Stroop interference effect, reflected in increased reaction times for incongruent stimuli as compared to congruent stimuli, will be greater during the depressed mood.

The electrophysiological correlates of the Stroop task are associated with activation within the 300-500ms post-stimulus time bin (West & Alain, 1999; Liotti et al., 2000), which reflects the localisation of context processing to the dorsolateral prefrontal cortex (George, Ketter, Parekh, Rosinsky, & Ring, 1994). However, additional processing of incongruent stimuli is reflected in a late lateralised positive complex, which has been localised to the temporo-parietal region (Liotti et al, 2000). Therefore the distributed cortical activation associated with the performance of the Stroop task may counteract the resource allocation problems. Consequently, an interaction with mood effects is not predicted. Davidson's model (Davidson, 1998a) implicates asymmetric frontal activation during the experience of negative mood, with hypoactivity within the left hemisphere. It is hypothesised that an induced negative mood will lead to altered activity recorded over frontal regions, which will be reflected in a modulation of frontal asymmetry.

## **6.2 Methods**

### **6.2.1 Participants**

Nine participants, 5 female and 4 male, with an average age of 26.2 years. All participants gave signed consent prior to taking part in the study and they were paid for their participation. All participants had achieved a score of 9 or less on the Beck Depression Inventory (BDI, (Beck, 1987). One participant did not pass the screening procedure for the study as they scored more than 9 on the BDI. As this represents mild to moderate depression (Beck, 1987), it was deemed inappropriate to include them in a mood manipulation study. All participants who failed to pass the selection criteria fully debriefed and informed that as their BDI score indicated they were feeling mildly unhappy it would be inappropriate to manipulate their mood.

### **6.2.2 Measures and Induction Procedures**

Two measures were used to assess mood levels during this study, the Beck Depression Inventory (BDI; Beck, 1987) and the Positive Affect and Negative Affect Schedule scale (PANAS; Watson et al., 1988).

The BDI consists of 21 symptoms and attitudes, subdivided into four or five evaluative statements, which are designed to assess the severity of depression in adolescents and adults (Beck, 1987). It is widely accepted that the BDI is able to detect possible depression in normal populations (Steer et al., 1985), therefore it has been used to screen participants prior to undergoing the mood induction procedure.

The PANAS consists of 20 adjectives which are designed to produce measures of Positive Affect (PA) and Negative Affect (NA) (Watson et al., 1988). The scales have been shown to be a reliable, valid and efficient method of assessing PA and NA (Watson et al., 1988).

The Velten mood induction procedure was used to produce a neutral and depressed mood. The use of this procedure is described in detail in Chapter 4, and the statements and instructions used within the neutral and depressed treatments are presented in Appendix 2.

### **6.2.3 Stimuli, Design and Procedure**

A repeated measure design was employed, where participants performed a Stroop task under a neutral and depressed condition. The experimental procedure followed during each condition was identical. Upon completion of the depressed condition, participants took part in the elation treatment from the Velten mood induction procedure (see Appendix 2) in order to reverse any remaining depressed mood.

Within a session, each participant completed two experimental blocks, each containing 72 trials (36 congruent, 36 incongruent). The stimuli consisted of the words 'red', 'blue', 'green' and 'yellow' in congruent and incongruent ink colours. There were 36 congruent stimuli where the colour word presented in the same ink colour and 36 incongruent stimuli where the colour word was presented in the other three colours (each of the three incongruent ink colours were used 3 times). The presentation of the congruent and incongruent trials was randomised across the block to ensure that participants would not detect any pattern of presentation. Furthermore, the randomisation would minimise any response priming or inhibition that may occur between trials.

Following the application of the Geodesic Sensor Net, participants completed a mood induction treatment and a Stroop task. Performance of the neutral and depressed mood induction treatments was counterbalanced across the participants. A mood assessment was performed prior to induction, following induction and at the end of the Stroop task. A key-colour-acquisition block was completed (36 trials) prior to the Stroop Task to help the participant learn the association between the response keys and colours. During the Stroop task, participants were instructed to press the colour key which responded to the colour of the ink in which words were presented on the screen.

Presentation of the mood induction statements and the Stroop task stimuli was controlled by the E-Prime experimental control system (Psychological Software Tools). During each trial a fixation cross was centrally presented for 200ms, then the stimuli were presented in the centre of the screen for 150ms. The inter-trial-interval randomly varied between 1700ms and 2200ms to ensure that the participant was not able to anticipate the beginning of each trial and therefore prevent any anticipatory activity within the ERP. Statements and

stimuli were presented upon a RM PC-5200 Professional Multimedia computer, with a 15-inch screen.

#### **6.2.4 EEG/ERP methods**

Scalp voltages were collected with a 128-channel Geodesic Sensor Net™ (Tucker, 1993), connected to a AC-coupled, 128-channel high input impedance amplifier (200M $\Omega$ , Net Amps™, Electrical Geodesics, Eugene, OR). Amplified analogue voltages (0.1Hz lowpass; 100 Hz highpass) were digitised at 250 Hz. Individual sensors were adjusted until impedances were less than 30 K $\Omega$ .

Trials were rejected from analysis if they contained more than 5 bad channels (changing more than 100  $\mu$ V between samples, or reaching amplitudes over 200  $\mu$ V). A moving window detection algorithm (NetStation™, Electrical Geodesics, Eugene, OR) marked channels containing vertical or horizontal eye movements (EOG channel differences greater than 70  $\mu$ V). A digital 30 Hz low pass filter was applied to the EEG recording prior to segmentation.

ERPs were computed within four different categories: neutral congruent, neutral incongruent, depressed congruent, depressed incongruent. Table 6.1 shows the number of trials included in the ERP for each participant. It can be seen that data from one participant was rejected due to the presence of too few observations to form reliable ERPs.

Participant	Neutral congruent	Neutral incongruent	Depressed congruent	Depressed incongruent
1	143	133	142	135
2	123	128	138	140
3*	79	83	140	133
4	126	111	128	113
5	137	141	138	136
6*	90	112	34	40
7*	35	31	114	126
8	122	127	140	136
9	115	110	121	113

**Table 6.1** The number of trials included within the ERPs calculated for the neutral congruent, neutral incongruent, depressed congruent and depressed congruent conditions for each participant.

An eye movement correction algorithm (Technical Appendix 2) was applied to trials contaminated with eye movements. All signal processing was conducted with a purpose-built signal processing toolbox for MatLab (MathWorks, Inc) (Technical Appendix 3). ERPs were baseline corrected with respect to a 100ms pre-stimulus recording interval. An average-rereference transformation was used to minimize the effects of reference site activity (Curran et al., 2003).

Using a modification of the regions of interest defined by (Curran et al., 1993b), ERPs were spatially average across the left frontal, left central, left parietal, left temporal, left occipital, right frontal, right central, right parietal, right temporal, and right occipital regions of interest. Sensors included in each region are detailed in Technical Appendix 3.

### **6.2.5 Statistical Analysis**

Scores from the negative affect and positive affect sub-scales of the PANAS were analysed using a repeated measures analysis with mood induction procedure (neutral, depressed) and test time (pre-induction, post-induction, post task) included as factors.

The behavioural data were analysed using a repeated measures analysis, with mood (neutral, depressed) included as the factor. Reaction times associated with correct response to congruent and incongruent stimuli will be included in the analysis.

The peak amplitude of event-related waveform components were analysed using a repeated measures analysis, with mood (neutral, depressed), stimulus type (congruent, incongruent), hemisphere (left, right), region of interest (frontal, central, parietal, occipital) and time bin (300-500, 500-700) included as factors.

## **6.3 Results**

### **6.3.1 Mood induction procedure**

Both the negative affect and positive affect sub-scales were analysed using a repeated measures analysis with mood induction procedure (neutral, depressed) and test time (pre-induction, post-induction, post task) included as factors. Where significant effects were found, the relationship between levels was investigated using a Bonferroni corrected pairwise comparison.



6.3.1.1 Negative affect

The estimated groups means and standard errors for the negative affect scores during each mood induction procedure are presented in Table 6.2. The difference in negative affect during the two mood induction procedures was not significant ( $F(1,8) = 0.03, p = > 0.05$ ).

Mood induction	Mean (N = 9)	SE
Neutral	12.63	1.15
Depressed	12.74	0.74

**Table 6.2** Estimated group means and standard errors (SE) calculated for negative affect scores under the neutral and depressed mood inductions.

The estimated groups means and standard errors for the negative affect scores at each time point are presented in Table 6.3. The difference in negative affect over the course of the experimental session was not significant ( $F(2,16) = 0.23, p = >0.05$ ).

Time point	Mean (N = 9)	SE
Pre-induction	13.11	1.060
Post-induction	12.61	0.923
Post-task	12.33	1.372

**Table 6.3** Estimated group means and standard errors (SE) calculated for negative affect scores at the three time points during the experimental session.

The interaction between mood induction procedure and test time was not significant ( $F(2,16) = 1.164, p = >0.05$ ), the estimated group means and standard errors for which are presented in Table 6.4.

Time point	Neutral Induction		Depressed induction	
	Mean (N = 7)	SE	Mean (N = 7)	SE
Pre-induction	13.44	1.63	12.78	0.83
Post-induction	11.89	0.90	13.33	1.09
Post-task	12.56	1.73	12.11	1.05

**Table 6.4** Estimated group means and standard errors (SE) calculated for negative affect scores at the three time points during the experimental session for each induction procedure.

### 6.3.1.2 Positive affect

The estimated groups means and standard errors for the positive affect scores during each mood induction procedure are presented in Table 6.5. The difference in positive affect during the two mood induction procedures was not significant ( $F(1,8) = 0.362$ ,  $p = > 0.05$ ).

Mood induction	Mean (N = 9)	SE
Neutral	30.78	2.67
Depressed	31.40	2.42

**Table 6.5** Estimated group means and standard errors (SE) calculated for positive affect scores under the neutral and depressed mood inductions.

The estimated group means and standard errors for positive affect at each time point are presented in Table 6.6. There was a significant main effect of test time ( $F(2,16) = 5.195$ ,  $p = 0.01$ ), which reflected a decrease in positive affect as the experimental session progressed. A post hoc test of this main effect did not reveal any significant pairwise differences.

Time point	Mean (N = 9)	SE
Pre-induction	33.50	1.94
Post-induction	31.78	2.26
Post-task	28.00	3.56

**Table 6.6** Estimated group means and standard errors (SE) calculated for positive affect scores at the three time points during the experimental session.

The interaction between mood and test time was not significant ( $F(2,16) = 2.37, p = > 0.05$ ), the estimated group means and standard errors for which are presented in Table 6.7

Time point	Neutral Induction		Depressed induction	
	Mean (N = 7)	SE	Mean (N = 7)	SE
Pre-induction	34.11	2.02	32.88	2.20
Post-induction	31.67	2.35	31.89	2.41
Post-task	26.56	4.09	29.44	3.12

**Table 6.7** Estimated group means and standard errors (SE) calculated for positive affect scores at the three time points during the experimental session for each induction procedure.

### 6.3.2 Behavioural data

All data were analysed using a repeated measures analysis, with mood (neutral, depressed) and stimulus type (congruent, incongruent) included as factors. Greenhouse-Geisser corrections were applied to results where assumptions of sphericity were violated. Where significant effects were found, pairwise comparisons were used to investigate the relationship between levels.

The mean reaction times for correct responses to congruent and incongruent stimuli are presented in Table 6.8. There was a significant main effect of stimulus type ( $F(1,8) = 59.684, p = < 0.001$ ). Reactions times to congruent stimuli were quicker than reaction times to incongruent stimuli.

Stimulus type	Mean (N = 9)	SE
Congruent	573.40	51.63
Incongruent	717.75	60.84

**Table 6.8** Mean reaction times (ms) and standard errors (SE) for congruent and incongruent stimuli.

Table 6.9 shows the correct reaction times to congruent and incongruent stimuli under the neutral and depressed mood conditions. There was no significant interaction between these two factors ( $F(1,8) = 0.278, p = > 0.05$ ).

Mood induction	Stimulus type	Mean (N = 9)	SE
Neutral	Congruent	585.48	53.37
	Incongruent	724.78	60.34
Depressed	Congruent	561.31	51.69
	Incongruent	710.69	64.74

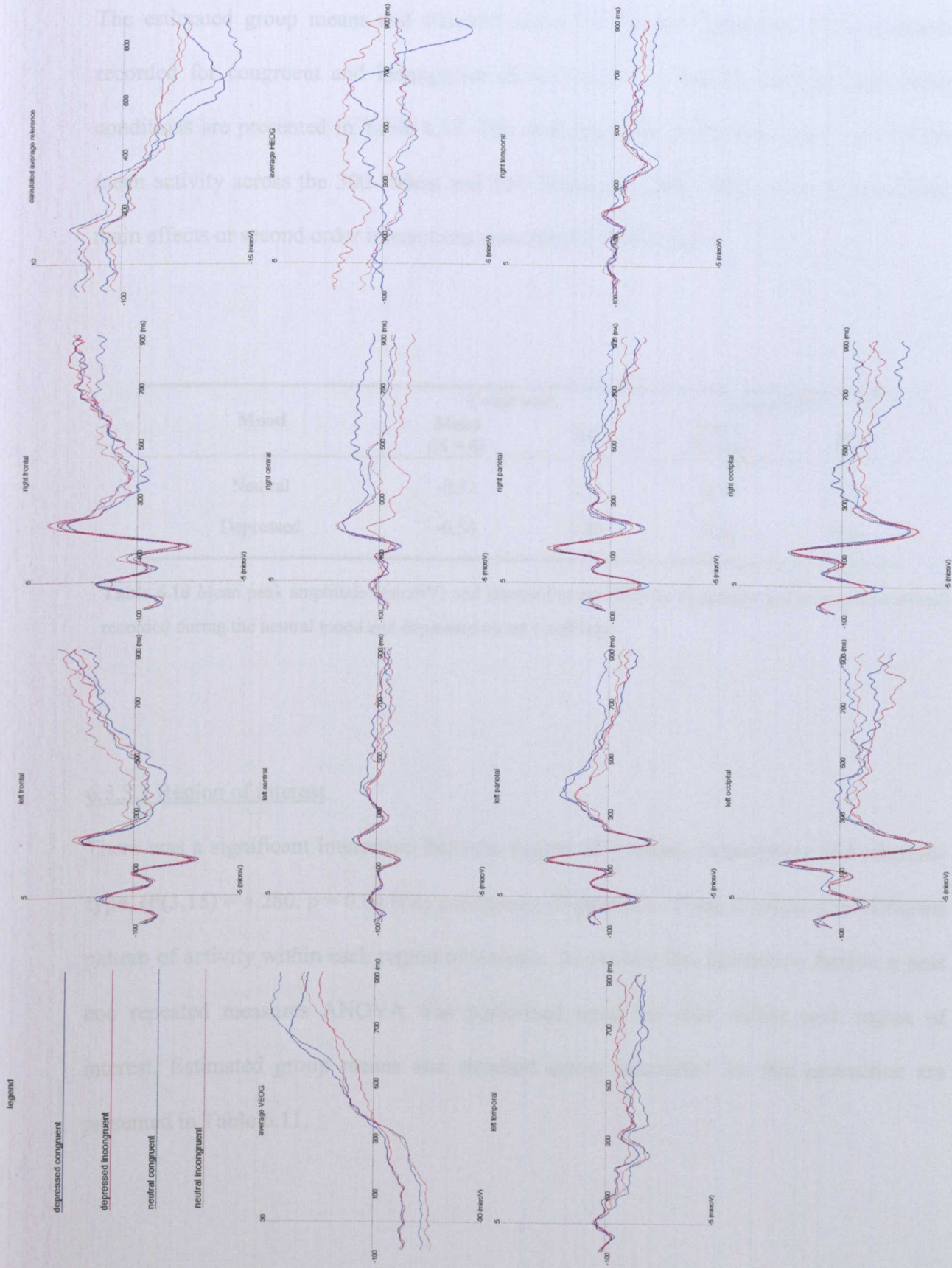
**Table 6.9** Mean reaction times (ms) and standard errors (SE) for congruent and incongruent stimuli under neutral and depressed moods.

### **3.3.3 Electrophysiological Data**

Following signal processing, 3 participants were excluded from analysis due to artifacts which remained in event-related potential waveforms. Figure 6.1 shows the grand averaged event-related potentials (6 participants) for congruent and incongruent stimuli recorded under the neutral and depressed mood conditions. Each graph represents the activity recorded for 100 ms prior to stimulus presentation and 900 ms after stimulus presentation. A similar distinction between congruent and incongruent waveforms was recorded over the left parietal area under both the neutral and depressed condition. However this distinction is not reflected within the other regions of interest. In the latter stages of the waveforms, from around 400ms, the maximum deflections over the frontal regions have a negative polarity and a positive polarity in more posterior regions.

The time of maximal activation was noted within the grand average ERPs and then checked within the individual average ERPs to ensure consistency across participants. Consequently, the peak amplitude of the maximum deflection within the 300-500ms and 500-700ms time bins was determined using an algorithm within the purpose-built signal processing toolbox (Technical Appendix 3).

Peak amplitude of waveform components were analysed using a repeated measures analysis, with mood (neutral, depressed), stimulus type (congruent, incongruent), hemisphere (left, right), region of interest (frontal, central, parietal, occipital) and time bin (300-500, 500-700) included as factors. Greenhouse-Geisser (GG) corrections were applied to results where assumptions of sphericity were violated. Where significant effects were found, the relationship between levels was investigated using a Bonferroni corrected pairwise comparison.



**Figure 6.1** The grand averaged event-related potentials for congruent and incongruent stimuli recorded under the neutral and depressed mood

### 6.3.3.1 Stimulus type

The estimated group means and standard errors for the peak amplitude of components recorded for congruent and incongruent stimuli under the neutral and depressed mood conditions are presented in Table 6.10. The data presented in the table below reflects the mean activity across the 300-500ms and 500-700ms time bins. There were no significant main effects or second order interactions associated with this factor.

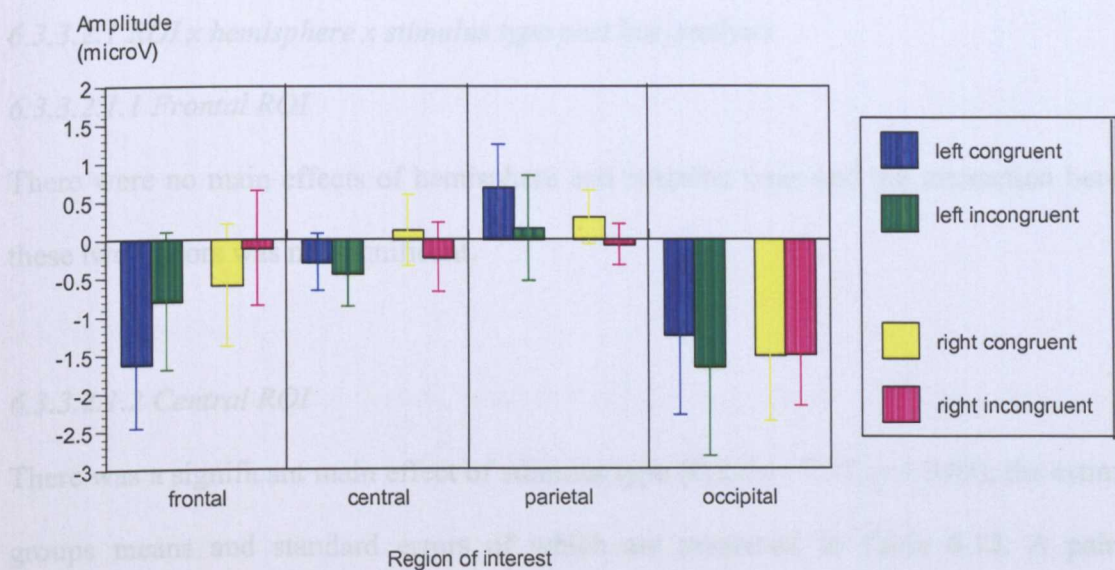
Mood	Congruent		Incongruent	
	Mean (N = 6)	SE	Mean (N = 6)	SE
Neutral	-0.51	0.14	-0.55	0.11
Depressed	-0.54	0.09	-0.59	0.13

**Table 6.10** Mean peak amplitude (microV) and standard errors (SE) for congruent and incongruent stimuli recorded during the neutral mood and depressed mood conditions.

### 6.3.3.2 Region of interest

There was a significant interaction between **region of interest**, **hemisphere** and **stimulus type**, ( $F(3,15) = 4.280$ ,  $p = 0.04$  (GG corrected)) (Figure 6.2). There is evidence of different pattern of activity within each region of interest. To explore this interaction further, a post hoc repeated measures ANOVA was performed upon the data within each region of interest. Estimated group means and standard errors calculated for this interaction are presented in Table 6.11.





**Figure 6.2** The mean peak amplitude (microV) recorded for congruent and incongruent stimuli within the left and right hemispheres of the regions of interest.

		Frontal		Central		Parietal		Occipital	
		Mean (N = 6)	SE	Mean (N = 6)	SE	Mean (N = 6)	SE	Mean (N = 6)	SE
Congruent	Left	-1.62	0.84	-0.29	0.38	0.68	0.57	-1.25	1.05
	Right	-0.59	0.80	0.12	0.47	0.29	0.35	-1.52	0.86
Incongruent	Left	-0.798	0.89	-0.45	0.42	0.15	0.69	-1.67	1.17
	Right	-0.11	0.75	-0.23	0.45	-0.07	0.27	-1.50	0.68

**Table 6.11** Mean peak amplitude (microV) and standard errors (SE) calculated for congruent and incongruent stimuli within the left and right hemispheres of the regions of interest.



#### *6.3.3.2.1 ROI x hemisphere x stimulus type post hoc analysis*

##### *6.3.3.2.1.1 Frontal ROI*

There were no main effects of hemisphere and stimulus type, and the interaction between these two factors was not significant.

##### *6.3.3.2.1.2 Central ROI*

There was a significant main effect of stimulus type ( $F(1,5) = 7.72$ ,  $p = 0.03$ ), the estimated groups means and standard errors of which are presented in Table 6.12. A pairwise comparison revealed that the mean difference between the peak amplitude recorded for congruent and incongruent stimuli was significant at the 0.05 level (Bonferroni corrected). There was greater negativity associated with incongruent stimuli than with congruent stimuli. The main effect of hemisphere and the interaction between hemisphere and stimulus type were not significant.

Stimulus type	Mean (N = 9)	SE
Congruent	-0.08	0.402
Incongruent	-0.341	0.403

**Table 6.12** Mean peak amplitude (microV) and standard errors (SE) calculated for congruent and incongruent stimuli within central region of interest.

##### *6.3.3.2.1.2.3 Parietal ROI*

There were no main effects of hemisphere and stimulus type, and the interaction between these two factors was not significant.

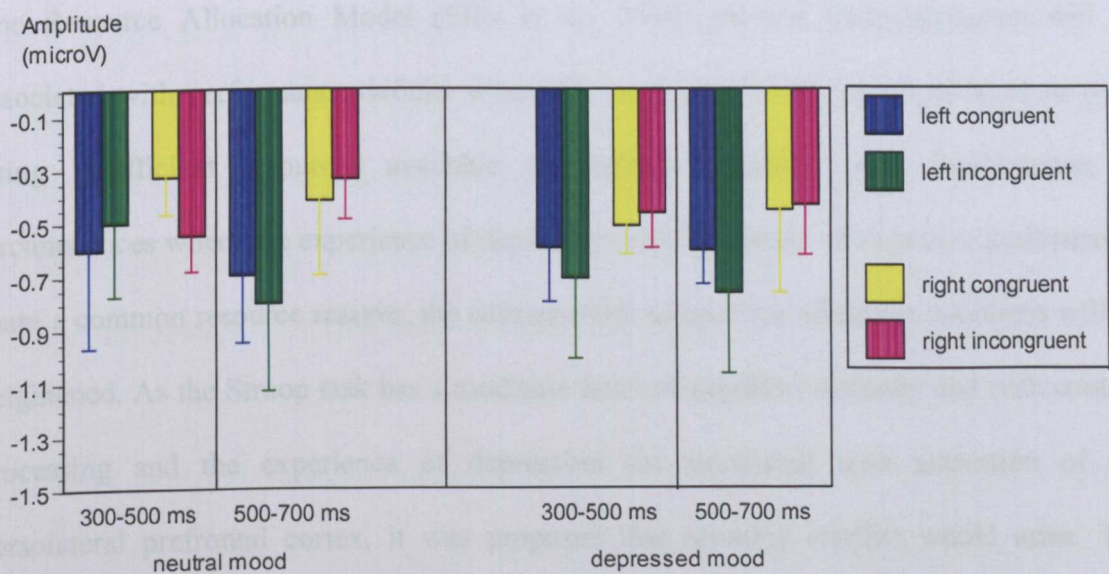
6.3.3.2.1.2.4 Occipital ROI

There were no main effects of hemisphere and stimulus type, and the interaction between these two factors was not significant.

6.3.3.3 Mood

The interaction between mood, stimulus type, hemisphere and time only neared significance ( $F(1,5) = 5.373, p = 0.06$ ) (Figure 6.3). Within the early time bin, the negative activity under the neutral condition is greater in reaction to congruent than to incongruent stimuli within the left hemisphere and greater in reaction to incongruent than congruent stimuli within the right hemisphere. Under the depressed mood condition, the pattern of activity across hemispheres in reaction to the congruent and incongruent stimuli is reversed. Within the later time bin this reversal of hemispheric asymmetry does not occur. To investigate this interaction further, a post hoc repeated measures ANOVA was performed upon the data within the early section of the waveform only.

6.4 Discussion



**Figure 6.3** The mean peak amplitude (microV) recorded for congruent and incongruent stimuli within the left and right hemispheres during the 300-500ms and 500-700ms time bins.

#### 6.3.3.3.1 Mood x stimulus type x hemisphere x time post hoc analysis

##### 6.3.3.3.1.1 Mood

The critical interaction between mood, stimulus type and hemisphere was not significant ( $F(1,5) = 1.253$ ,  $p = > 0.05$ ), estimated group means and standard errors for the interaction are presented in Table 6.13.

Mood	Hemisphere	Congruent		Incongruent	
		Mean (N = 6)	SE	Mean (N = 6)	SE
Neutral	Left	-0.60	0.37	-0.49	0.28
	Right	-0.33	0.11	-0.55	0.14
Depressed	Left	-0.59	0.21	-0.71	0.31
	Right	-0.51	0.11	-0.46	0.14

**Table 6.13** Mean peak amplitude (microV) and standard errors (SE) calculated for congruent and incongruent stimuli within left and right hemispheres under the neutral and depressed mood conditions.

## 6.4 Discussion

The Resource Allocation Model (Ellis et al., 1988) predicts that depression will be associated with performance deficits when the experience of the emotions leads to there being insufficient resources available to perform cognitive task. Furthermore, in circumstances where the experience of depression and the control of cognitive performance share a common resource reserve, the susceptibility to resource allocation problems will be heightened. As the Stroop task has a moderate level of cognitive intensity and both context processing and the experience of depression are associated with activation of the dorsolateral prefrontal cortex, it was proposed that resource conflict would arise. The normal Stroop effect, where congruent stimuli are processed faster than incongruent, will be attenuated by induced negative mood, with increased reaction times and/or error rates.

Early frontal ERPs, associated with lateral PFC and ACC activation (Liotti et al, 2000) will be altered in negative mood, with a reduction in amplitude of negative potentials in the depressed mood condition.

#### **6.4.1 Mood Induction Procedure**

The mood profile expected during a depressed mood induction was of no change in negative affect scores and a decrease in positive affect scores on the PANAS. This would reflect the individual maintaining their state of negative affect and not becoming more anxious or tense, but experiencing a decrease in their positive state, representing an increase in sadness. Neutral mood induction would be associated with no change in either negative or positive affect scores on the PANAS, thereby reflecting maintenance of the individual's baseline mood state.

Under both conditions, there was no difference between the assessments of negative affect performed prior to and following the mood induction. The levels of negative affect did not change at the end of the experimental session. Therefore people maintained a steady level of negative affect throughout the experimental session, reflecting no alterations in the levels of anxiety and tension they were experiencing. This is an important point as the Stroop task is notoriously frustrating to perform and the maintenance of negative affect shows task performance itself did not interfere with the mood induction.

There was no significant effect of mood induction upon positive affect, therefore similar levels of positive affect were experienced under the neutral and depressed mood induction procedures. The significant effect of time indicated that there was a decrease in positive affect over the experimental session. This reflected an increase in sadness experienced by

the participants across the experimental session. However this increase was a gradual effect as the pairwise comparison did not reveal any significant mean differences.

The lack of an interaction between the mood induction and test time reflects that the same pattern of reduction was associated with both the neutral and depressed induction procedures. Participants experienced an initial increase in sadness after completing the mood induction procedure, and then reported a further increase at the end of the experimental session.

In terms of negative affect, the appropriate mood profiles were not created by the induction procedures. Neither the neutral or depression treatment altered the level of anxiety and tension experienced by the participants. However, the increase in sadness experienced during both the neutral and depressed induction procedures means that the mood profile associated with both conditions was the same. Therefore all mood related effects reported in this study must be viewed in light of this lack of subjective difference between mood states.

#### **6.4.2 Behavioural Data**

The Stroop interference effect was successfully demonstrated in this study, with significantly slower reaction times recorded for incongruent stimuli in comparison to congruent stimuli ( $p = 0.001$ ). However the first hypothesis was not supported as the strength of the interference effect was not greater under the depressed mood, with participants slower at responding to incongruent than to congruent stimuli under both mood conditions.

There are two possible explanations for this result. In light of the unsuccessful mood induction, it is possible that this result simply reflects the consistent performance of the participants on two separate occasions. However, as the sensitivity of the subjective mood assessment is under question, there is the possibility that behavioural performance is not susceptible to the influence of mood because of compensatory neuronal activation. The assessment of the electrophysiological data allows this interpretation to be investigated.

#### **6.4.3 Electrophysiological Data.**

Before the influence of mood can be assessed, the electrophysiological differentiation between congruent and incongruent stimuli must be considered. Previous studies have indicated that incongruent stimuli are associated with an increase in negativity over frontal regions (Liotti et al., 2000; West et al., 1999). There was a significant interaction between region of interest, hemisphere and stimulus type ( $p = 0.04$ ). In Figure 6.2, it can be seen that the negative amplitudes recorded over the frontal regions of interest showed a tendency towards asymmetry in response to congruent and incongruent stimuli. This was not borne out in analysis as there were no significant main effects or interactions within this region of interest.

There was a significant main effect of stimulus type ( $p = 0.03$ ) within the central region of interest, where incongruent stimuli were associated with greater degree of negative activation. This topography is consistent with West & Alain (1999), who reported an increase in negativity towards incongruent stimuli over fronto-central regions. It should be noted that most of the studies reporting specifically frontal effects employed modified versions of the Stroop task (Zysset et al, 2001, Milham et al, 2001). No other effects were found in the parietal and occipital regions. Liotti et al's (2000) finding of a lateralised late

positive complex over the left temporo-parietal region in the 500-800 ms time window in responses to incongruent stimuli was not replicated in this study.

There was no influence of mood upon negative activity solely within the frontal region of interest. This result is consistent with the apparent failure of the mood induction procedure to manipulate mood as shown by the PANAS results. However, the verbal reports of sadness during the depressed condition do suggest that participants experienced a sad mood. Therefore, with respect to potential changes in asymmetry that would be predicted from Davidson's model, it is worth noting the interaction between mood, stimulus type, hemisphere and time that neared significance ( $p = 0.06$ ).

This is illustrated in Figure 6.3, where an interesting relationship between activation within the 300-500ms time bin can be seen. Previous studies have reported lateralisation to the left temporo-parietal region in the latter stages of ERP, which are thought to reflect semantic processing of the incongruent stimuli (Liotti et al., 2000), but none have reported earlier asymmetries. In this study, within the 300-500ms time bin, the negative activity under the neutral condition is greater for congruent than incongruent stimuli within the left hemisphere and greater for incongruent than congruent stimuli within the right hemisphere. In the depressed mood condition, this pattern of activity across hemispheres in reaction to the congruent and incongruent stimuli is reversed. This change in asymmetry is associated with the relative stability in the size of the left hemisphere asymmetry in the two conditions, as compared to a substantially reduced right hemisphere asymmetry in the depressed condition.

In summary, the Stroop effect was effectively demonstrated at the behavioural and partly at the cortical level with incongruent stimuli associated with greater negative activation in central regions. At the subjective and behavioural level, the mood manipulation was not effective, but there was tentative evidence of cortical activation being modulated by the two mood conditions. As in the previous study, there was a reduction in asymmetrical activation in the depressed condition, in this case associated with asymmetries on the right hemisphere. Such reductions in asymmetry could be associated with an increase in activation to produce the same level of task performance, a form of compensatory recruitment.

As before, the mood manipulation was apparently not effective as measured by the subjective mood assessment scale, although the task performance itself was associated with a more sustained mood change. The task employed in this study was chosen because of its increased cognitive demands and greater reliance on frontal control. There were differences at the cortical level between responses to congruent and incongruent stimuli, but they were not specifically focussed in the frontal areas, although the Stroop task employed here was not modified as in other studies (Zysset et al, 2001, Milham et al, 2001). At the cortical level, there is still a failure to find mood-related frontal changes, although there were differences in overall cortical asymmetries in the different mood conditions. Again this lack of focus could be related both to the apparent failure to induce mood change and to the lower level of task demand. In the next study, a task associated with a higher level of cognitive complexity will be chosen, again designed to more specifically 'target' frontal areas of control.



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## **Chapter 7**

### **Mood and Cognition III – The influence of depressed mood on a sustained attention task**

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#### **7.1 Introduction**

Sustained attention tasks can be seen as having a high level of cognitive intensity. In addition to stimulus identification and context processing, task-relevant information must be available for assessment and manipulation to ensure successful performance. Neuroanatomical activation within sustained attention tasks has also been located to the dorsolateral prefrontal cortex (Smith & Jonides, 1997). As this cortical region is implicated in the experience of depression, and the tasks have a high level of cognitive intensity, it is proposed that sustained attention tasks will be susceptible to the detrimental effects of depressed mood. To allow the electrophysiological investigation of this hypothesis, an n-back working memory task (Gray, 1999) will be performed under neutral and depressed moods.

##### **7.1.1 Working memory**

Working memory is responsible for the storage and manipulation of information on a temporary basis, and therefore underlies the majority of our cognitive abilities (Tagamets et al., 2000). The dominant hypothesis describes it as a multi-component system which

includes two modality specific slave systems, the phonological loop and visuospatial sketch pad, and the central executive (Baddeley, 1986). The components have been localised within the prefrontal cortex (Smith & Jonides, 1999), and although they are functionally related, evidence from neuropsychological patients suggests they can be dissociated (D'Esposito & Postle, 2000).

Smith & Jonides (1999) proposed the processes controlled by the components of working memory to include:

- *Attention and inhibition* – the focusing of attention onto relevant information within the environment and associated processes and the active inhibition of irrelevant information and processes;
- *Task management* – scheduling information processing during complex tasks when attention must be switched between different task components;
- *Planning* – formulation of sequence routines when a number of subtasks must be completed during goal accomplishment;
- *Storage* - the active maintenance of task-relevant information within working memory;
- *Monitoring* – assessing and updating the contents of working memory in order to determine the progress through a sequential task;
- *Coding* – assigning temporal and spatial information to working memory representations.

Working memory is seen as a self-contained system as the accomplishment of these processes is achieved under constrained conditions, both in terms of the storage capacity and available resources (Baddeley, 1986; Baddeley & Hitch, 1974; Richardson, 1996). The

notion of working memory being a resource controlled system is the basic premise of the major opponent to Baddeley and colleagues' multi-modular theory. Just & Carpenter (1992) postulate that working memory comprises two limited capacity systems, one specializing in spatial working memory and the other on verbal working memory. Within this model, all aspects of processing of spatial and verbal information are performed by the appropriate self-contained working memory system.

One of the significant ways in which Baddeley's model of working memory has been developed is the structure of the slave systems which sub-serve the executive processes. Rather than there being a single visual-based information slave system, it is now proposed that distinct object and spatial stores exist (Smith et al., 1997).

#### **7.1.2 Methodological constraints of working memory studies in humans**

There are a number of limitations which surround the investigation of the neuroanatomical correlates of working memory in humans. It is only under rare circumstances, such as evaluation procedures during epilepsy surgery (Amador & Fried, 2004), that single-cell recording can be conducted on human participants. Furthermore, neuropsychological studies in humans are restricted by the fact that few naturally occurring lesions are anatomically discrete (Pierrot-Deseilligny, Rivaud, Gaymard, & Agid, 1991; Ptito, Crane, Leonard, Amsel, & Caramanos, 1995; Verin et al., 1993). The use of neuroimaging techniques, both haemodynamic and electromagnetic, allows the spatial and temporal dynamics of brain activity to be assessed during the performance of working memory tasks (D'Esposito et al., 1998a).

### 7.1.2.1 Working Memory Tasks

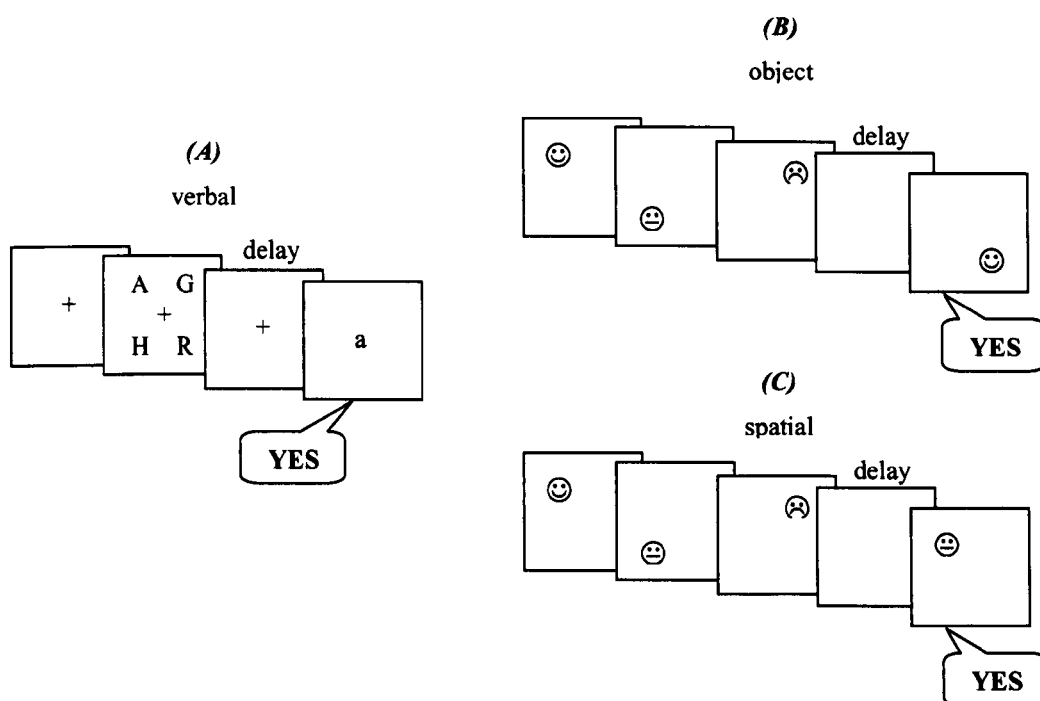
There are four main working memory tasks employed in neuroimaging studies, which can be seen to vary upon the following dimensions:

- task modality – the task stimuli can be verbal, spatial or object;
- cognitive complexity – a hierarchy of complexity can be created by varying the number of actions to be performed upon information being held within working memory;
- cognitive workload – increasing levels of workload can be achieved by varying the amount of information held in working memory.

#### *7.1.2.1.1 Item recognition task*

Verbal, object and spatial examples of the item recognition task have been widely utilised in working memory research (Smith, Jonides, Marshuetz, & Koeppel, 1998) and is a modification of the delayed object recognition tasks used in primate research. In the verbal paradigm (Figure 7.1 A), the participant is presented with a small array of target letters and, after a delay period, a single probe letter. The aim of the participant is to decide whether the probe matches any of the target letters. In the object condition (Figure 7.1 B) three different faces are presented sequentially in different locations and then, after an interval, a probe face is presented. The aim of the participant is to indicate whether the probe face is the same one of the target faces. The spatial condition (Figure 7.1 C) uses the same stimuli, but the decision centres on whether the probe position matches any of the target stimuli positions.

Although the workload involved can be manipulated by varying the number of items within the target array/sequence, the item recognition task is associated with a low cognitive



**Figure 7.1** The trial structure of the item recognition task within the (A) verbal, (B) object, and (C) spatial modalities. Adapted from Smith & Jonides (1999).

complexity. The target items must be stored within working memory however because the task is performed on a trial-by-trial basis, there is no monitoring or coding involved as once a decision has been made a new set of target items enters working memory.

#### 7.1.2.1.2 Letter-span task

During this task the participant is presented with an array of letters and the aim is to either remember them in the order they were presented or to alphabetise them (D'Esposito, Postle, Ballard, & Lease, 1999). The cognitive workload associated with this task can be manipulated by increasing the number of letters presented in the array. A medium level of cognitive complexity is associated with the task as the letters must be stored and manipulated within working memory, however the trial-by-trial basis of array presentation means there is no continual up-date of information.

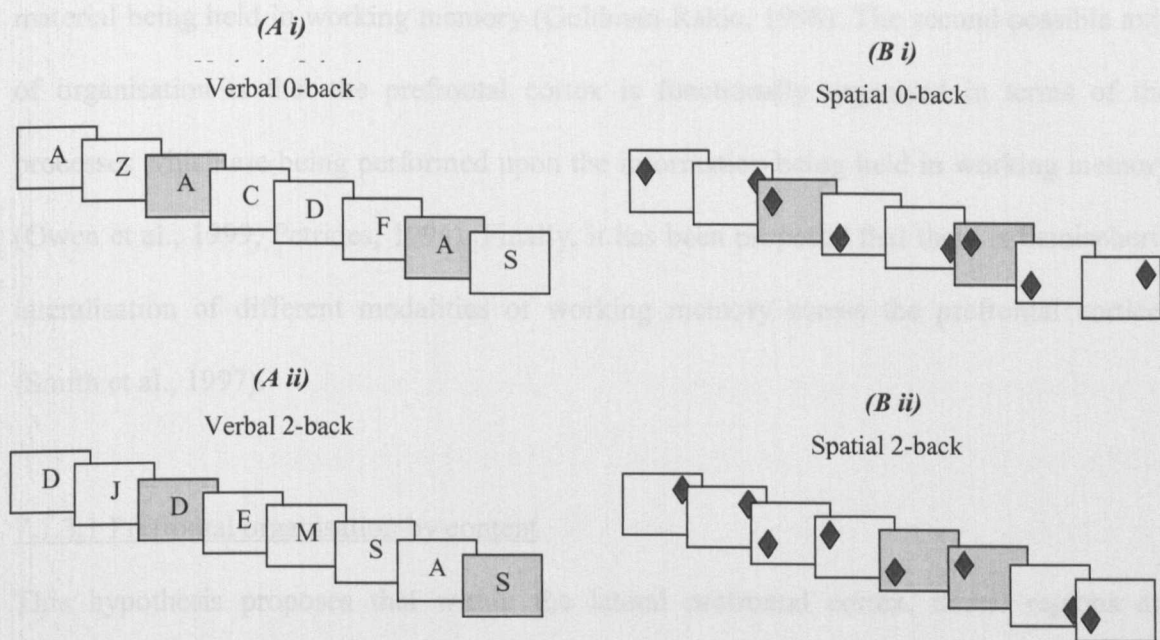
#### *7.1.2.1.3 Running memory task*

Within this task, participants are presented with lists which vary in item length (Kusak, Grune, Hagendorf, & Metz, 2000). During each trial the items are presented sequentially, and the participant's aim is to recognise or recall the three most recent items at the end of each trial. Cognitive workload can be manipulated by increasing the list size, and in trials containing more than three items complexity is increased as the stored information must be coded and updated. Although this produces a cognitively intense task, the trial-by-trial nature of the task means data stored within working memory do not require continual updating.

#### *7.1.2.1.4 N-back task*

The n-back working memory task (Jonides et al., 1997), allows the full manipulation of cognitive workload and complexity levels. In the verbal 0-back task (Figure 7.2 Ai), the participant is presented with a series of letters and is instructed to remember the first letter of the series. The participants must then indicate whether each letter in the series is the same or different to the target letter. Workload can be increased by altering the number of letters over which a comparison must be made.

For example, in the 2-back version of the task (Figure 7.2 Aii), the participant must compare each letter in the series to that which was presented two trials previously and indicate whether it is the same or different from the continually changing target letter. In the spatial version of this task (Figure 7.2 Bi and ii), the same instructions apply, but the participant must focus upon the location of the stimulus rather than the stimulus itself.



**Figure 7.2** The task structure of the n-back working memory task within the (A) verbal and (B) spatial, modalities. Example of low workload (i) and high workload (ii) are given for both modality. Adapted from D’Esposito et al. (1993).

Within the low workload example of the n-back task, storage and monitoring of the information contained within working memory must be conducted for successful completion of the task. However, when cognitive workload is manipulated by increasing the number of items which must be stored in working memory, so too is the cognitive complexity of the task. Not only must the information be stored and monitored, it must also be coded for its temporal position in the sequence and this information must be continually up-dated as the target stimuli change throughout the experimental session.

### **7.1.3 Neuroanatomical correlates**

There are three main hypotheses surrounding the functionality of the prefrontal cortex in humans. The first follows from the postulations surrounding the functional organisation of the prefrontal cortex in primates (Wilson, Scallidhe, & Goldman-Rakic, 1993b), stating that the dorsolateral and ventrolateral regions are functionally distinct in terms of the type of

material being held in working memory (Goldman-Rakic, 1998). The second possible axis of organisation is that the prefrontal cortex is functionally organised in terms of the processes which are being performed upon the information being held in working memory (Owen et al., 1999; Petrides, 1994). Finally, it has been proposed that there is hemispheric lateralisation of different modalities of working memory across the prefrontal cortices (Smith et al., 1997).

#### 7.1.3.1 Prefrontal organisation by content

This hypothesis proposes that within the lateral prefrontal cortex, dorsal regions are responsible for spatial processing while ventral regions are responsible for object/non-spatial processes (Haxby, Ungerleider, Horwitz, Rapoport, & Grady, 1995; Courtney, Ungerleider, Keil, & Haxby, 1996). Aside from its consistency with single-cell recordings in primates (Wilson, Scallidhe, & Goldman-Rakic, 1993a; Scallidhe, Wilson, & Goldman-Rakic, 1999; Goldman-Rakic, 1996), support for this hypothesis came from neurophysiological evidence such as the robust connections that exist between the lateral prefrontal cortex and the ventral visual pathway (Ungerleider, Gaffan, & Pelak, 1989; Webster, Bachevalier, & Ungerleider, 1994).

However, in a comparative analysis of spatial and non-spatial neuroimaging studies the localisation of prefrontal activation was found to be mixed (D'Esposito et al., 1998b). D'Esposito et al (1998) cited evidence of a distributed pattern of activation across the prefrontal cortex in both spatial and non-spatial tasks when data were plotted on to a standardised brain (McCarthy et al., 1996; Smith, Jonides, & Koeppe, 1996b; Verin et al., 1993). Furthermore, in their own empirical study, evidence was provided of activation



within the dorsolateral prefrontal cortex in both spatial and non-spatial working memory (D'Esposito et al., 1998b).

#### 7.1.3.2 Prefrontal organisation by process

The postulated distribution of processes across the prefrontal cortex can be interpreted as being modulated by cognitive complexity. The 'two-stage model' proposes that there are two executive functioning processes located within the lateral prefrontal cortex (Owen, Evans, & Petrides, 1996; Petrides, 1994). The ventral region is thought to be responsible for the active storage of information within working memory, while the higher intensity processes, such as manipulation and coding, are thought to be controlled by the dorsal region (Owen et al., 1996).

There are a number of neuroimaging studies which provide support for this hypothesis. For example, a PET study compared activation during a running memory task and the memory-only condition of a letter-span task (Salmon et al., 1996), therefore creating a cognitive complexity difference in terms of the up-dating of information held within working memory. It was discovered that the dorsolateral prefrontal cortex was associated with greater activation during the running memory task when it was compared with the letter-span task. During the lower complexity letter-span task, activation was associated with the ventrolateral prefrontal cortex alone (Salmon et al., 1996).

Furthermore, there is evidence of there being differential levels of activation which reflect degrees of cognitive complexity. In order to differentiate between the maintenance and manipulation of information, an event-related fMRI study compared brain activation during the memory and manipulation conditions of a five character version of the letter-span task

was employed (D'Esposito et al., 1999). Greater activation levels within the dorsolateral prefrontal cortex were associated with the manipulation of the letters during the delay period (D'Esposito et al., 1999).

During their comparative study, D'Esposito et al (1998) grouped together those studies which employed tasks that required only the maintenance of information and compared activation patterns to those associated with 'maintenance plus' tasks (this classification covered tasks which involved manipulation, within-trial up-dating and across trial up-dating of information stored within working memory). A clear distinction between the regions of the prefrontal cortex was discovered, with maintenance-only tasks being associated with ventral activation and maintenance-plus tasks being associated with dorsal activation (D'Esposito et al., 1998b).

#### 7.1.3.3 Lateralisation of function

Finally it has been proposed that there is hemispheric specialisation for spatial versus non-spatial (object/verbal) working memory (Smith et al., 1997). Using a PET paradigm, a clear double dissociation was found between activation of the right dorsolateral prefrontal cortex during spatial and the left dorsolateral prefrontal cortex during verbal working memory tasks (Smith, Jonides, & Koeppe, 1996a). In more recent studies however, this clear distinction between the two hemispheres has not been replicated (Nystrom, Braver, Sabb, & et al, 2000; Postle, Stern, Rosen, & et al, 2000).

One explanation for this inconsistency is that the lateralisation of visual working memory is task-context specific, as it has been proposed that effects are modulated by memory set size, retention period length and item familiarity (Ungerleider, Courtney, & Haxby, 1998).

Alternatively, specific processes within working memory could be lateralised. Using a item-recognition task, the process of matching the target probe with the information held within working memory was found to be lateralised to the left hemisphere (Talsma, Wijers, Klaver, & Mulder, 2001).

However, the existence of domain-specific findings in neuroimaging studies means the argument for hemispheric lateralisation is pervasive. For example, a shape sensitive region has been localised within the left prefrontal cortex (Nystrom et al., 2000) and a location sensitive regions has been isolated within the right prefrontal cortex (Zurowski, Gostomecyk, Groen, & et al, 2002). Consequently it is considered that hemispheric relative dominance for function, rather than specialisation is associated with left and right prefrontal regions (Walter et al., 2003).

#### **7.1.4 Electrophysiological studies of working memory**

##### **7.1.4.1 Event-related potentials and working memory**

Although electrophysiological techniques have been widely used in the investigation of memory processes (see reviews by Rugg, 1995; Basar, Basar-Eroglu, Karakas, & Schuermann, 2000), their utilization in working memory research is less extensive. There are a number of studies which investigate the way in which working memory processes constrain language processing (for example Kolk, Chwilla, van Herten, & Oor, 2003; Gunter, Wagner, & Friederici, 2003)), however less have used the technique to directly explore the temporal dynamics of working memory as a complex cognitive process.

Often, inferences as to the electrophysiological correlates of working memory have been made when the successful completion of the experimental task utilizes working memory

processes. For example, Rugg and colleagues concluded that verbal working memory was associated with slow waves lateralised to the left hemisphere through studies of phonological processing (Barrett & Rugg, 1990; Rugg, 1984b; Rugg, 1984a) and object working memory was reflected in slow waves lateralised to the right hemisphere through studies of face perception (Barrett & Rugg, 1989; Barrett, Rugg, & Perrett, 1988). This association with slow wave activity has been borne out in studies explicitly investigating working memory (Garcia-Larrea & Cezanne-Bert, 1998; Gevins et al., 1996; Kiss, Pizio, Francois, & Schoplocher, 1998; Ruchkin, Canoune, Johnson, & Ritter, 1995; Ruchkin, Johnson, Grafman, Canoune, & Ritter, 1992; Ruchkin, Johnson, Mahaffey, & Sutton, 1988; Ruchkin, Munsen, & Sutton, 1982).

Employing an item recognition task, Ruchkin et al (1992) found a frontal negativity which was thought to represent non-modality specific working memory processes, with a tentative conclusion that it reflected executive functions. Furthermore, the amplitude of the negative slow waves was believed to act as an index of the cognitive workload associated with the task, with increasing negativity reflecting higher workload. In a similar item recognition task, frontal negative activity was postulated to reflect general control processes supplemented by parieto-occipital negative activity associated with storage (Berti, Geissler, Lachman, & Mecklinger, 2000).

Ruchkin et al (1992) also reported that topographically distinct slow waves were elicited during the processing of phonological and visual-spatial information. This was therefore seen as providing evidence of modality specific slave systems within working memory (Ruchkin et al., 1992). As visual-spatial information is thought to be more efficiently processed within working memory (Logie, 1986), the topographical similarity within the

waveform was consistent with the view that there is a direct link between storage and maintenance. Alternatively, the more intensive processing of the verbal information was associated with a complex pattern of slow wave activation, with the presence of early temporo-occipital negativity, mid task parietal positivity, and late frontal negativity (Ruchkin et al., 1992),

As previously stated, trials in running memory tasks where lists increase in size from three items represents the point at which updating of information within working memory must occur. Using the recognition condition, a marked increase in positivity was seen when list size changed from three to four items, followed by smaller increments for subsequent increases in list size (Kiss et al., 1998). Kiss et al (1998) concluded that this parietal positivity reflected both updating and interference suppression processes.

Kusak et al (2000) took this scenario a stage further by employing the higher workload version of this running memory task, where the final three items must be recalled rather than recognised. Although their results were comparable to Kiss et al (1998) at early stages of information processing, the increment in cognitive difficulty was associated with a fronto-central positivity (Kusak et al., 2000). It was concluded that this component specifically represented the update of working memory involved with the recall version of the running memory task (Kusak et al., 2000).

During a more cognitively complex task, both verbal and spatial 3-back conditions were associated with a frontal positive component lateralised to the left hemisphere at around 450ms post-stimulus, which was thought to reflect the processes associated with the sustained attention required during the task (Gevins et al., 1996). This was seen to be

consistent with neuroimaging evidence of frontal activation associated with sustained attention to sequential presentation of stimuli (for example, Cohen et al., 1994; Sergent, Zuck, Levesque, & MacDonald, 1992).

#### 7.1.5.1 Investigating the influence of depression using event-related potentials

Although memory deficits are common in patients suffering from depression (Pelosi, Slade, Blumhardt, & Sharma, 2000), few studies have employed event-related potentials to investigate the influence of depression upon working memory. In an assessment of mood congruency effects, Deldin et al (2001) investigated whether a clinical population exhibited differences in slow wave activity in response to emotionally charged stimuli within a working memory task. It was postulated that slow wave activity would increase in negativity in response to negatively valenced stimuli within the depressed participants, while non-depressed individuals would show an increased negativity towards positively valenced stimuli (Deldin et al., 2001).

Deldin et al (2001) employed an affective adaptation of an item recognition task, where an emotional adjective (S1), which could have a positive, negative or neutral valency, was randomly paired with a letter (S2). The aim of the participant was to indicate whether the letter had occurred in the previously presented emotional word. The results showed depressed and non-depressed individuals differentially processed positive and negative information within a low-level working memory task.

Specifically, when the non-depressed individuals were holding information within working memory, positive information was associated with greater negative slow wave activity than negative information (Deldin et al., 2001). Alternatively, depressed individuals displayed

greater negative activity in response to the storage of negative information within working memory than was measured towards positive information (Deldin et al., 2001). It was concluded that this represented a more elaborate processing of mood congruent information during working memory tasks in both the depressed and non-depressed individuals (Deldin et al., 2001).

The second ERP study again employed a low cognitive intensity task, when performance on a item-recognition task with set sizes of one, three and five was compared in depressed patients and healthy controls (Pelosi et al., 2000). ERPs recorded from the depressed patients showed increased negative amplitudes within the 375-840 post-stimulus time period when compared to the healthy controls. It was concluded that the increase in negativity was associated with the recruitment of additional neuronal populations (Pelosi et al., 2000).

Within the same time period, there was a reduction in later positive activity recorded in the depressed patients. As previously stated, the cognitive correlates of late positive activity within ERPs are unclear, however the reduction in positivity was concluded to reflect abnormal working memory processes during the integration of information in the final stages of the task (Pelosi et al., 2000). Together, these negative and positive abnormalities were seen to reflect the dysfunctional control of timing and resource allocation by executive processes within depressed individuals (Pelosi et al., 2000).

### **7.1.5 The Resource Allocation Model and Working Memory**

As working memory is a closed energy system, situations where processing demand exceeds supply will lead to deterioration in cognitive performance reliant upon working

memory (Vos, Gunter, Kolk, & Mulder, 2001). For example verbal performance deficits are associated with: individuals exhibiting low memory spans, cognitively complex tasks and high cognitive workload (Just & Carpenter, 1992). Likewise, the resource allocation model predicts that depression will have a detrimental effect on performance in situations where there is insufficient resources to perform the task at hand (Ellis & Ashbrook, 1987).

In comparison to passive and selective attention tasks, working memory is a cognitively intense process which requires sustained attention. When working memory processes are employed in realistic settings and high workload and intensity paradigms such as the n-back task, correct performance is dependent upon the successful completion of a number of sub-goals by system components. It is presumed that the experience of a depressed mood will lead to there being insufficient resources to perform these sub-goals and consequently working memory performance will be affected.

#### **7.1.6 Experimental Hypothesis**

This study will employ an n-back working memory task as this allows the three dimensions of task modality, cognitive complexity, and cognitive workload to be altered. It is predicted that reaction times and percentage errors will be higher in the more cognitively demanding task. Given the involvement of frontal regions in working memory tasks (Smith et al., 1997), it is also assumed that behavioural indices of working memory performance will be sensitive to mood changes. It is hypothesised that the reaction times and percentage error rates associated with the high workload conditions of the working memory tasks will be increased under the depressed mood.



With respect to electrophysiological data, increasing task complexity is hypothesised to be associated with increasing frontal negativity (Ruchkin et al, 1992; Deldin et al, 2001) and with increased parietal positivity (Kiss et al, 1998).

The WM task employed here will also manipulate verbal and spatial aspects of task demands. It is hypothesised that there will be an asymmetry in frontal negativity, with left hemisphere activation being greater than right in the verbal task and right hemisphere activation being greater than left in the spatial task in early time windows (Smith and Jonides, 1997; Barrett and Rugg, 1990). These asymmetries will be reduced in the depressed mood condition (Pelosi et al, 2000).

## **7.2 Methods**

### **7.2.1 Participants**

Eight participants, 4 female and 4 male, with an average age of 22.4 years. All participants gave signed consent prior to taking part in the study and they were paid for their participation. All participants had achieved a score of 9 or less on the Beck Depression Inventory (BDI; Beck, 1987).

### **7.2.2 Measures and induction procedures**

Two measures were used to assess mood levels during this study, the Beck Depression Inventory (BDI; Beck, 1987) and the Positive Affect and Negative Affect Schedule scale (PANAS; Watson et al., 1988).

The BDI consists of 21 symptoms and attitudes, subdivided into four or five evaluative statements, which are designed to assess the severity of depression in adolescents and

adults (Beck, 1987). It is widely accepted that the BDI is able to detect possible depression in normal populations (Steer et al., 1985), therefore it has been used to screen participants prior to undergoing the mood induction procedure.

The PANAS consists of 20 adjectives which are designed to produce measures of Positive Affect (PA) and Negative Affect (NA) (Watson et al., 1988). The scales have been shown to be a reliable, valid and efficient method of assessing PA and NA (Watson et al., 1988).

The Velten mood induction procedure was used to produce a neutral and depressed mood. The use of this procedure is described in detail in Chapter 4, and the statements and instructions used within the neutral and depressed treatments are presented in Appendix 2.

### **7.2.3 Stimuli, design and procedure**

A repeated measure design was employed, where participants performed a working memory task under a neutral and depressed condition. The experimental procedure followed during each condition was identical. Upon completion of the depressed condition, participants took part in the elation treatment from the Velten mood induction procedure (see Appendix 2) in order to reverse any remaining depressed mood. Due to the increased time associated with performing this experiment, 5 Velten mood statements were presented to the participants in between performance of the 4 experimental blocks

Within a session, participants completed 2 control blocks (verbal 0-back, spatial 0-back), 2 training blocks (verbal 3-back, spatial 3-back) and 4 experimental blocks (2 verbal 3-back, 2 spatial 3-back). Control and experimental blocks contained 72 trials and training blocks

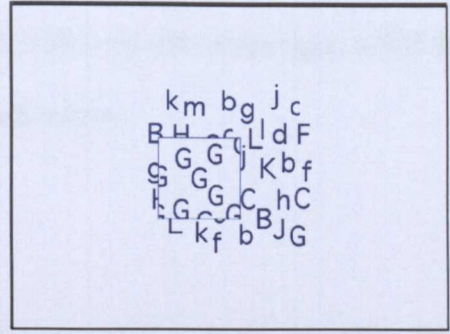
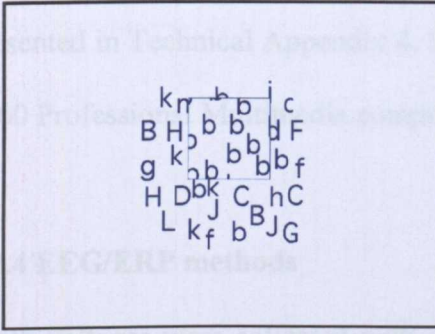
contained 36 trials. Presentation of stimulus type followed a pseudo-random sequence, and presentation of verbal and spatial blocks was counterbalanced across sessions.

Following the application of the Geodesic Sensor Net, participants completed a mood induction treatment and the working memory tasks. Performance of the neutral and depressed mood induction treatments was counterbalanced across the participants. A mood assessment was performed prior to induction, following induction and at the end of the working memory tasks.

In a similar design to that used by Gevins et al (1996), the same stimuli were utilised for spatial and verbal version of a 0-back and 3-back task. As the stimuli used in this study were positioned on a circular grid it is possible that spatial orientation cues were provided to participants. Therefore this study used stimuli developed by Gray (1999) where the stimulus were placed upon an array of letters (Figure 7.3), therefore removing any unintentional spatial cues (Gray, 1999; Lavric, Rippon, & Gray, 2003).

During the verbal 0-back condition, participants were told to focus upon the letters presented, regardless of their typeface and location upon the screen. The participants were instructed to remember the first letter presented in the block and compare this letter to each subsequent letter. They were instruction to press key '1' if the letters were the same and key '2' if the letters were different.

During the spatial 0-back condition, participants were told to focus upon the position of the letters presented, rather than the actual letters themselves. The participants were instructed to remember the position in which the first letter was presented in the block and compare



**Figure 7.3** Stimuli employed in the both the verbal and spatial variant of the 0-back and 3-back working memory task.

this position to the position of each subsequent letter. They were instruction to press key ‘1’ if the positions were the same and key ‘2’ if the positions were different.

During the verbal 3-back condition, participants were told to focus upon the letters presented, regardless of their typeface and location upon the screen. The participants were instructed to compare each letter presented to the letter which appeared three trials previously. They were instruction to press key ‘1’ if the letters were the same and key ‘2’ if the letters were different.

During the spatial 3-back condition, participants were told to focus upon the position of the letters presented, rather than the actual letters themselves. The participants were instructed to compare the position of each letter presented to the position of the letter which appeared three trials previously. They were instruction to press key ‘1’ if the letters were the same and key ‘2’ if the letters were different.

Presentation of the mood induction statements and the working memory stimuli was controlled by the E-Prime experimental control system (Psychological Software Tools). As this was a novel experimental paradigm, the development of the E-Prime protocol is

presented in Technical Appendix 4. Statements and stimuli were presented upon a RM PC-5200 Professional Multimedia computer, with a 15-inch screen.

#### **7.2.4 EEG/ERP methods**

Scalp voltages were collected with a 128-channel Geodesic Sensor Net™ (Tucker, 1993), connected to a AC-coupled, 128-channel high input impedance amplifier (200M $\Omega$ , Net Amps™, Electrical Geodesics, Eugene, OR). Amplified analogue voltages (0.1Hz lowpass; 100 Hz highpass) were digitised at 250 Hz. Individual sensors were adjusted until impedances were less than 30 K $\Omega$ .

Trials were rejected from analysis if they contained more than 5 bad channels (changing more than 100  $\mu$ V between samples, or reaching amplitudes over 200  $\mu$ V). A moving window detection algorithm (NetStation™, Electrical Geodesics, Eugene, OR) marked channels containing vertical or horizontal eye movements (EOG channel differences greater than 70  $\mu$ V). A digital 30 Hz low pass filter was applied to the EEG recording prior to segmentation.

ERPs were computed within eight different categories: neutral verbal 0-back, neutral spatial 0-back, neutral verbal 3-back, neutral spatial 3-back, depressed verbal 0-back, depressed spatial 0-back, depressed verbal 3-back, depressed spatial 3-back. No participants were rejected from the analysis. Table 7.1 shows the number of trials included in the ERP for each participant.

Participant	Neutral				Depressed			
	Verbal 0-back	Verbal 3-back	Spatial 0-back	Spatial 3-back	Verbal 0-back	Verbal 3-back	Spatial 0-back	Spatial 3-back
1	50	71	71	63	47	116	47	115
2	67	110	57	105	62	76	62	121
3	59	74	34	69	55	45	62	60
4	40	101	35	124	32	78	27	101
5	42	104	39	98	66	115	66	108
6	45	103	44	70	71	121	66	120
7	69	107	68	96	64	110	62	94
8	24	68	26	61	21	49	28	51

**Table 7.1** The number of trials included within the ERPs calculated for the neutral verbal 0-back, neutral verbal 3-back, neutral spatial 0-back, neutral spatial 3-back, depressed verbal 0-back, depressed verbal 3-back, depressed spatial 0-back and depressed spatial 3-back conditions for each participant.

An eye movement correction algorithm (Technical Appendix 2) was applied to trials contaminated with eye movements. All signal processing was conducted with a purpose-built signal processing toolbox for MatLab (MathWorks, Inc) (Technical Appendix 3). ERPs were baseline corrected with respect to a 100ms pre-stimulus recording interval. An average-rereference transformation was used to minimize the effects of reference site activity (Curran et al., 2003).

Using a modification of the regions of interest defined by (Curran et al., 1993b), ERPs were spatially average across the left frontal, left central, left parietal, left temporal, left occipital, right frontal, right central, right parietal, right temporal, and right occipital regions of interest. Sensors included in each region are detailed in Technical Appendix 3.

### **7.2.5 Statistical Analysis**

Scores from the negative affect and positive affect sub-scales of the PANAS were analysed using a repeated measures analysis with mood induction procedure (neutral, depressed) and test time (pre-induction, post-induction, post task) included as factors.

The behavioural data were analysed using a repeated measures analysis, with mood (neutral, depressed) included as the factor. Reaction times associated with correct response to congruent and incongruent stimuli will be included in the analysis.

The peak amplitude of event-related waveform components were analysed using a repeated measures analysis, with mood (neutral, depressed), workload (0-back, 3-back), modality (spatial, verbal), hemisphere (left, right), region of interest (frontal, central, parietal, occipital) and time bin (300-500, 500-700) included as factors.

## **7.3 Results**

### **7.3.1 Mood induction procedure**

Both the negative affect and positive affect sub-scales were analysed using a repeated measures analysis with mood induction procedure (neutral, depressed) and test time (pre-induction, post-induction, post task) included as factors. Where significant effects were found, the relationship between levels was investigated using a Bonferroni corrected pairwise comparison.

7.3.1.1 Negative affect

The estimated groups means and standard errors for the negative affect scores during each mood induction procedure are presented in Table 7.2. The difference in negative affect during the two mood induction procedures was not significant ( $F(1,7) = 1.770, p = > 0.05$ ).

Mood induction	Mean (N = 8)	SE
Neutral	13.29	1.58
Depressed	14.00	1.87

**Table 7.2** Estimated group means and standard errors (SE) calculated for negative affect scores under the neutral and depressed mood inductions.

The estimated groups means and standard errors for the negative affect scores at each time point are presented in Table 7.3. It can be seen that negative affect increases across the experimental session, however the main effect only approached significance ( $F(2,14) = 3.406, p = 0.06$ ).

Time point	Mean (N = 8)	SE
Pre-induction	12.81	1.79
Post-induction	13.69	1.74
Post-task	14.44	1.71

**Table 7.3** Estimated group means and standard errors (SE) calculated for negative affect scores at the three time points during the experimental session.

The interaction between mood induction procedure and test time was not significant ( $F(2,14) = 1.101, p = > 0.05$ ), the estimated group means and standard errors for which are presented in Table 7.4.



Time point	Neutral Induction		Depressed induction	
	Mean (N = 7)	SE	Mean (N = 7)	SE
Pre-induction	12.88	1.62	12.75	2.07
Post-induction	13.13	1.47	14.25	2.03
Post-task	13.88	1.73	15.00	1.74

**Table 7.4** Estimated group means and standard errors (SE) calculated for negative affect scores at the three time points during the experimental session for each induction procedure.

**7.3.1.2 Positive affect**

The estimated groups means and standard errors for the positive affect scores during each mood induction procedure are presented in Table 7.5. The difference in positive affect during the two mood induction procedures was not significant ( $F(1,7) = 0.318, p = > 0.05$ ).

Mood induction	Mean (N = 8)	SE
Neutral	23.67	2.66
Depressed	24.63	2.09

**Table 7.5** Estimated group means and standard errors (SE) calculated for positive affect scores under the neutral and depressed mood inductions.

The estimated group means and standard errors for positive affect at each time point are presented in Table 7.6. There was a significant main effect of test time ( $F(2,14) = 26.52, p = < 0.001$ ), which reflected a decrease in positive affect as the experimental session progressed. A pairwise comparison revealed that the mean reduction in positive affect between pre-induction and post-induction was significant ( $p = 0.008$ ), the mean reduction in positive affect between post-induction and post-task was significant ( $p = 0.01$ ) and the reduction in positive affect between pre-induction and post-task was significant ( $p = 0.002$ ).

Time point	Mean (N = 8)	SE
Pre-induction	27.81	2.29
Post-induction	24.00	2.29
Post-task	20.63	2.34

**Table 7.6** Estimated group means and standard errors (SE) calculated for positive affect scores at the three time points during the experimental session.

The interaction between mood and test time neared significance ( $F(2,14) = 3.37, p = 0.06$ ), the estimated group means and standard errors for which are presented in Table 7.7. There is a decrease in positive affect associated with both mood induction procedures, which continues until the post-task mood assessment.

Time point	Neutral Induction		Depressed induction	
	Mean (N = 7)	SE	Mean (N = 7)	SE
Pre-induction	26.38	2.71	29.25	2.29
Post-induction	23.63	2.81	24.38	2.22
Post-task	21.00	2.78	20.25	2.05

**Table 7.7** Estimated group means and standard errors (SE) calculated for positive affect scores at the three time points during the experimental session for each induction procedure.

### 7.3.2 Behavioural data

Percentage error rates and correct response reaction times were analysed using a repeated measures analysis, with mood (neutral, depressed), workload (0-back, 3-back) and modality (spatial, verbal) included as factors. Greenhouse-Geisser corrections were applied to results where assumptions of sphericity were violated. Where significant effects were found, the

relationship between levels was investigated using a Bonferroni corrected pairwise comparison.

7.3.2.1 Percentage error rate analysis

The estimated group means and standard errors for percentage error rates during the two workload levels under neutral and depressed moods are presented in Table 7.8. As no significant main effects or interactions were found within the percentage error rate analysis, no other results will be reported

Mood	0-back		3-back	
	Mean (N = 8)	SE	Mean (N = 8)	SE
Neutral	20.9	9.23	24.99	6.07
Depressed	27.03	7.89	22.103	4.57

**Table 7.8** Estimated group means (%error) and standard errors (SE) calculated for the 0-back and 3-back workload levels during the neutral and depressed conditions.

7.3.2.2 Reaction time analysis

*7.3.2.2.1 Mood*

The mean reaction times (ms) and standard errors for correct responses under neutral mood and depressed mood conditions are presented in Table 7.9. Although reaction times were slower during the depressed condition than during the neutral condition, this difference was not significant ( $F(1,7) = 0.586, p = > 0.05$ ).

Mood induction	Mean (N = 8)	SE
Neutral	621.00	94.01
Depressed	680.97	61.31

**Table 7.9** Mean reaction times (ms) and standard errors (SE) for correct responses during the neutral mood and depressed mood conditions.

#### 7.3.2.2.2 Workload

The mean reactions times (ms) and standard errors for correct responses associated with the 0-back and 3-back workload conditions are presented in Table 7.10. Reaction times during the 3-back version of the working memory task were significantly slower than those during the 0-back version ( $F(1,7) = 11.147, p = 0.01$ ).

Workload	Mean (N = 8)	Standard Error
0 back	621.52	63.37
3 back	680.46	75.29

**Table 7.10** Mean reaction times (ms) and standard errors (SE) calculated for correct responses during 0-back and 3-back workload conditions.

#### 7.3.2.2.3 Modality

The mean reactions times (ms) and standard errors for correct responses associated with the spatial and verbal conditions are presented in Table 7.11. Reaction times during the verbal version of the working memory task were significantly slower than those during the spatial version ( $F(1,7) = 5.449, p = 0.05$ ).

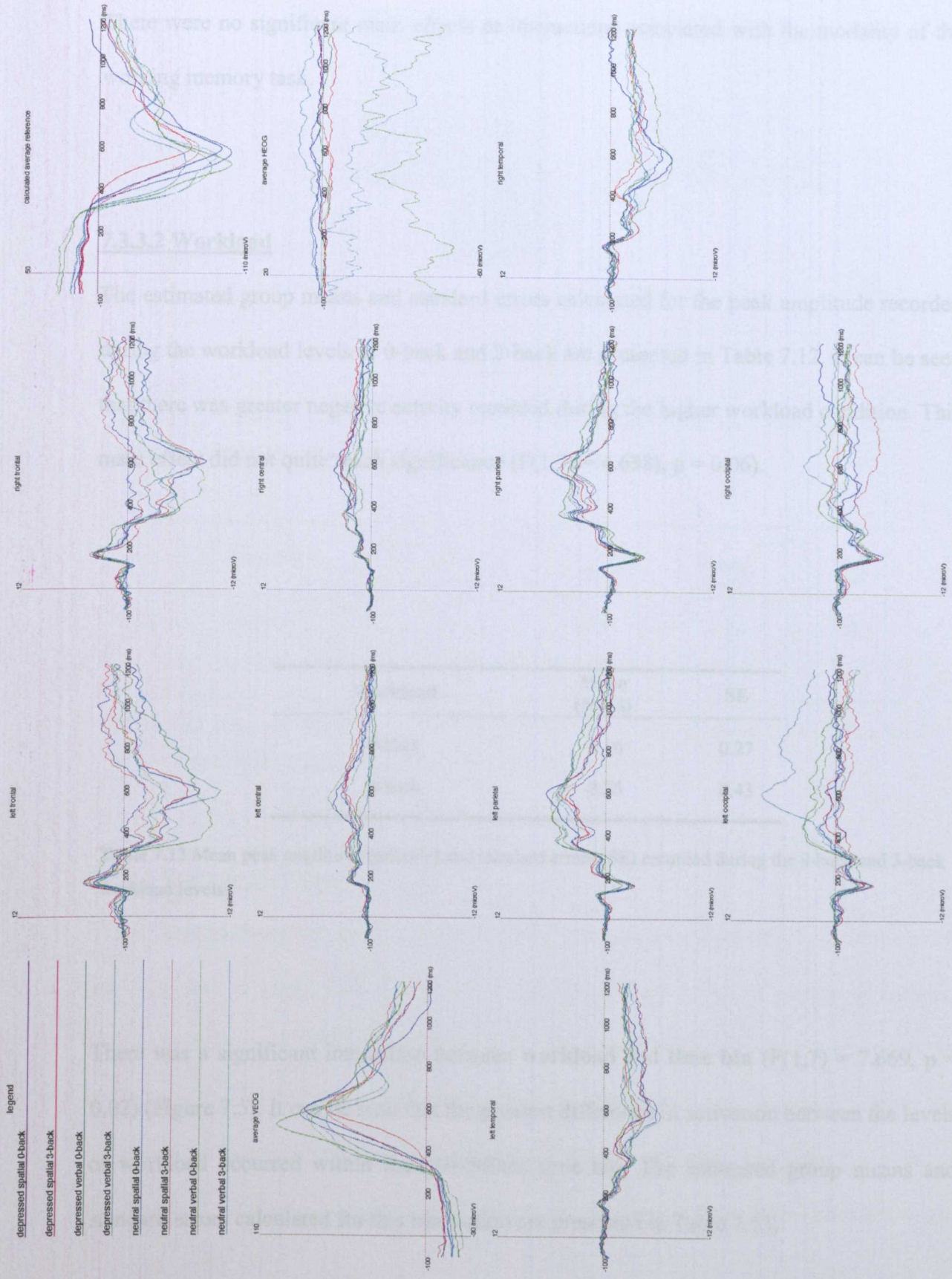
Task modality	Mean (N = 8)	Standard Error
Spatial	543.27	106.55
Verbal	758.71	49.33

**Table 7.11** Mean reaction times (ms) and standard errors (SE) calculated for correct responses during the spatial and verbal conditions.

### 7.3.3 Electrophysiological data

Figure 7.4 shows the grand averaged event-related potentials for verbal and spatial trials under both workload conditions recorded under neutral and depressed moods. Each graph represents the activity recorded for 100 ms prior to stimulus presentation and 1200 ms after stimulus presentation. The time of maximal activation was noted within the grand average ERPs and then checked within the individual average ERPs to ensure consistency across participants. Consequently, the peak amplitude of the maximum deflection within the 300-500ms and 500-700ms time bins was determined using an algorithm within the purpose-built signal processing toolbox (Technical Appendix 3).

Peak amplitude of waveform components were analysed using a repeated measures analysis, with mood (neutral, depressed), workload (0-back, 3-back), modality (spatial, verbal), hemisphere (left, right), region of interest (frontal, central, parietal, occipital) and time bin (300-500, 500-700) included as factors. Greenhouse-Geisser (GG) corrections were applied to results where assumptions of sphericity were violated. Where significant effects were found, the relationship between levels was investigated using a Bonferroni corrected pairwise comparison.



**Figure 7.4** The grand averaged event-related potentials for verbal and spatial trials at 0-back and 3-back workload levels, recorded under the neutral and depressed mood conditions.

### **7.3.3.1 Modality**

There were no significant main effects or interactions associated with the modality of the working memory task.

### **7.3.3.2 Workload**

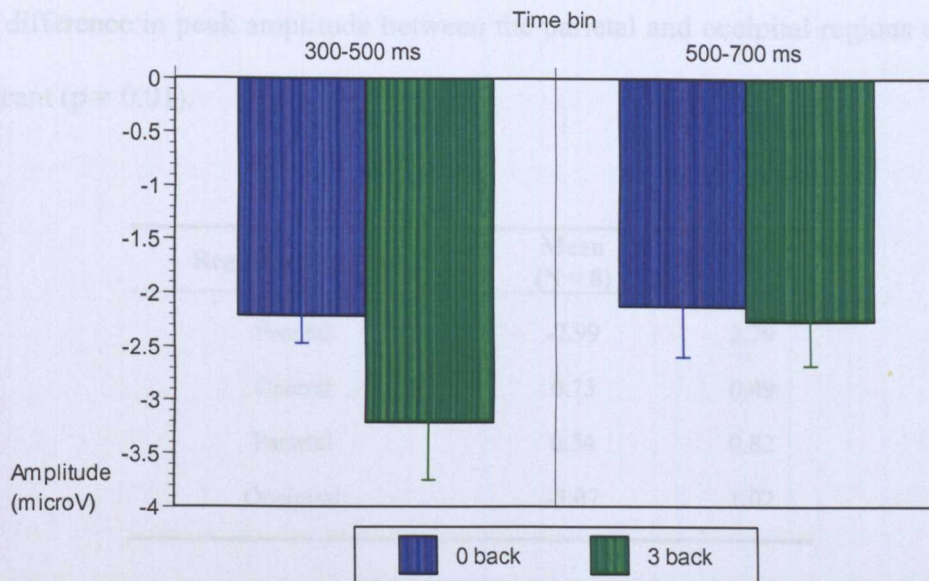
The estimated group means and standard errors calculated for the peak amplitude recorded during the workload levels of 0-back and 3-back are presented in Table 7.12. It can be seen that there was greater negative activity recorded during the higher workload condition. This main effect did not quite reach significance ( $F(1,7) = 4.638$ ),  $p = 0.06$ ).

<b>Workload</b>	<b>Mean (N = 8)</b>	<b>SE</b>
0-back	-2.16	0.27
3-back	-2.73	0.43

**Table 7.12** Mean peak amplitude (microV) and standard errors (SE) recorded during the 0-back and 3-back workload levels.

There was a significant interaction between **workload** and **time bin** ( $F(1,7) = 7.669$ ,  $p = 0.02$ ) (Figure 7.5). It can be seen that the greatest difference in activation between the levels of workload occurred within the 300-500ms time bin. The estimated group means and standard errors calculated for this interaction are presented in Table 7.13.





**Figure 7.5** The mean peak amplitude (microV) recorded in the 0-back and 3-back workload levels during the 300-500ms and 500-700ms time bin.

Time bin	0-back		3-back	
	Mean (N = 8)	SE	Mean (N = 8)	SE
300-500 ms	-2.21	0.26	-3.20	0.55
500-600 ms	-2.12	0.48	-2.26	0.42

**Table 7.13** Mean peak amplitude (microV) and standard errors (SE) calculated for the 0-back and 3-back workload levels during the 300-500ms and 500-700ms time bins.

### 7.3.3.3 Regions of interest

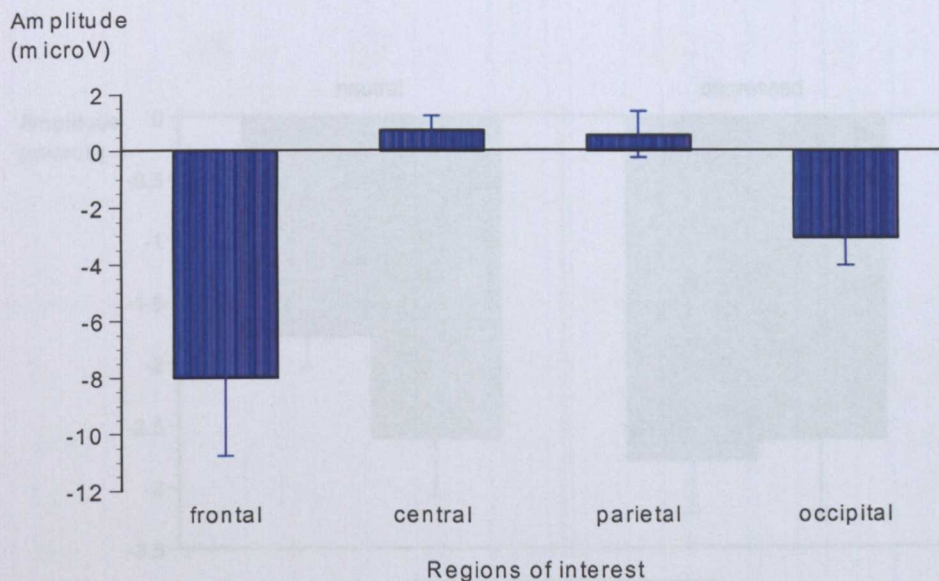
The estimated group means and standard errors calculated for the four regions of interest are presented in Table 7.14. There was a significant main effect of **region of interest** ( $F(3,21) = 5.392, p = 0.007$ ) (Figure 7.6). It can be seen that peak activity varies across the scalp with the frontal and occipital regions being associated with negativity, while the central and parietal regions are associated with little activity at all. There is also variation in the degree of activation, however a pairwise comparison of mean difference shows that



only the difference in peak amplitude between the parietal and occipital regions of interest is significant ( $p = 0.01$ ).

Region of Interest		Mean (N = 8)	SE
Hemisphere	Frontal	-7.99	2.79
	Central	0.73	0.49
	Parietal	0.54	0.82
	Occipital	-3.07	1.02

**Table 7.14** Mean peak amplitude (microV) and standard errors (SE) calculated for the four regions of interest.



**Figure 7.6** The mean peak amplitude (microV) recorded over the four regions of interest.

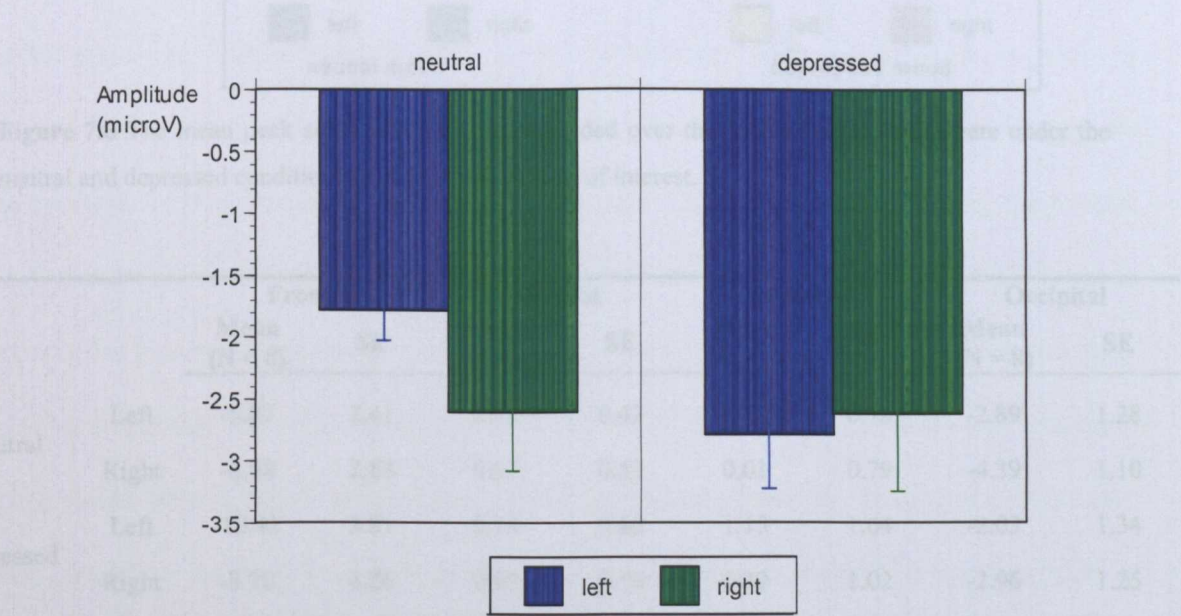
#### **7.3.3.4 Mood**

The estimated group means and standard errors calculated for the peak amplitude in the left and right hemispheres under the neutral and depressed moods are presented in Table 7.15. It can be seen that the peak negative amplitude of activation with during the depressed condition is greater than during the neutral condition. Furthermore the difference in peak

amplitude between the left and right hemispheres is reduced during the depressed condition. Unfortunately this interaction did not reach significance ( $F(1,7) = 3.910$ ,  $p = 0.08$ ) (Figure 7.7), however it is worth noting in light of the third order interaction.

Hemisphere	Neutral		Depressed	
	Mean (N = 8)	SE	Mean (N = 8)	SE
Left	-1.78	0.23	-2.79	0.44
Right	-2.61	0.48	-2.62	0.64

**Table 7.15** Mean peak amplitude (microV) and standard errors (SE) calculated for the left and right hemisphere within the neutral and depressed conditions

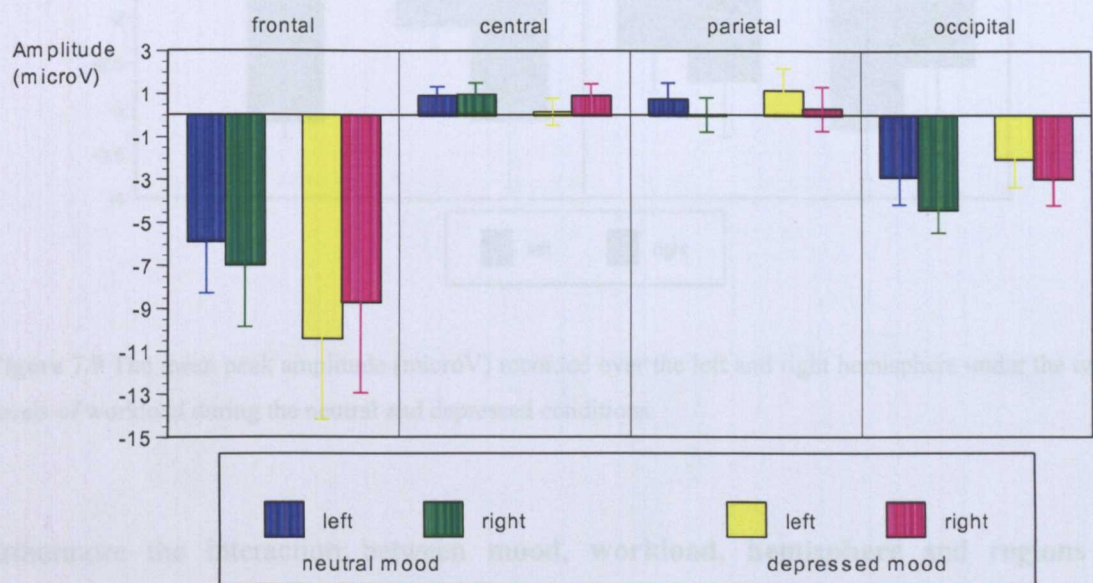


**Figure 7.7** The mean peak amplitude (microV) recorded over the left and right hemisphere within the neutral and depressed conditions.

There was a significant interaction between **mood**, **hemisphere** and **region of interest** ( $F(3,21) = 3.635$ ,  $p = 0.03$  (GG corrected)) (Figure 7.8). It can be seen that the largest amplitude response occurs within the frontal region of interest and that mood differentially hemisphere differences between the workload levels during the neutral mood are reduced during the depressed mood.



modulates the laterality of activation. The group estimated means and standard errors calculated for this interaction are presented in Table 7.16.

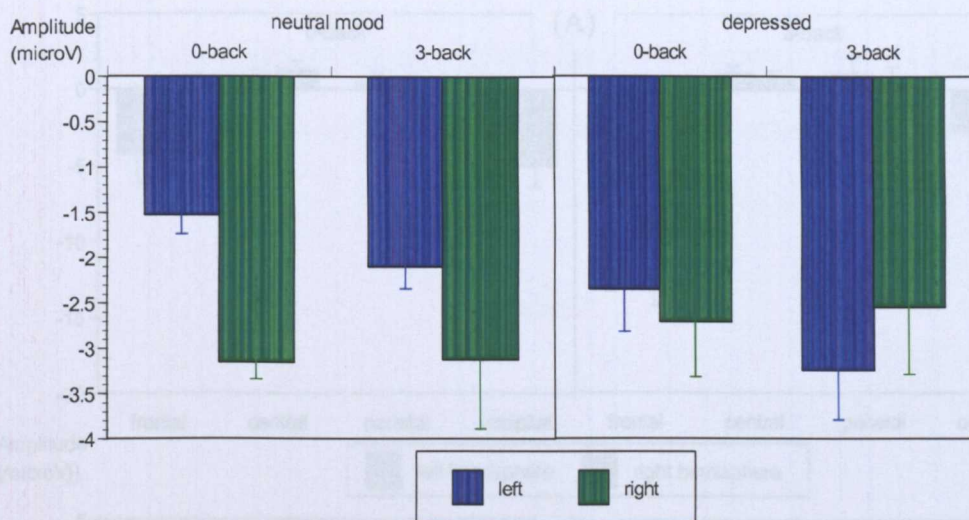


**Figure 7.8** The mean peak amplitude (microV) recorded over the left and right hemisphere under the neutral and depressed conditions in each of the regions of interest.

		Frontal		Central		Parietal		Occipital	
		Mean (N = 8)	SE	Mean (N = 8)	SE	Mean (N = 8)	SE	Mean (N = 8)	SE
Neutral	Left	-5.87	2.41	0.92	0.42	0.73	0.78	-2.89	1.28
	Right	-6.98	2.88	0.97	0.53	0.01	0.79	-4.39	1.10
Depressed	Left	-10.41	3.81	0.14	0.62	1.15	1.04	-2.03	1.34
	Right	-8.70	4.26	0.89	0.56	0.29	1.02	-2.96	1.25

**Table 7.16** Mean peak amplitude (microV) and standard errors (SE) calculated for the left and right hemisphere under the neutral and depressed conditions, within each region of interest.

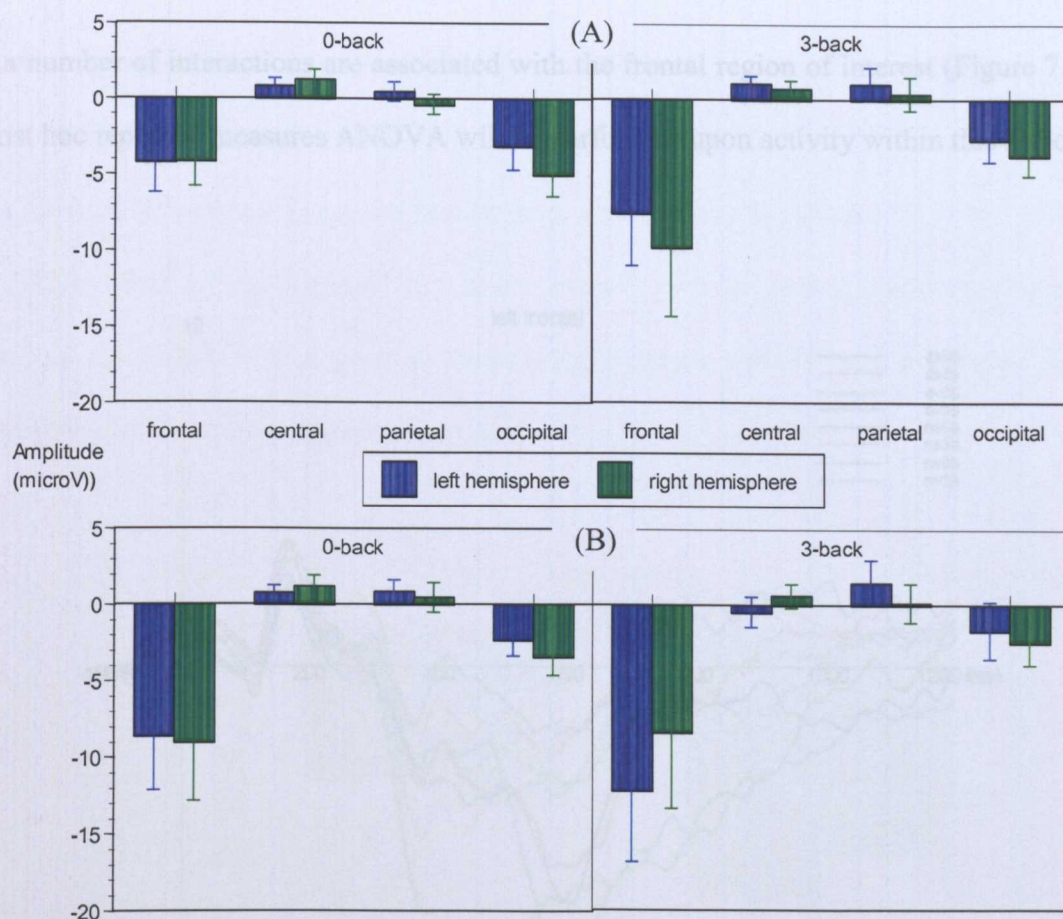
It is also interesting to note that the interaction between mood, hemisphere and workload also neared significance ( $F(1,7) = 3.931, p = 0.08$ ) (Figure 7.9). It can be seen that hemispheric differences between the workload levels during the neutral mood are reduced during the depressed condition.



**Figure 7.9** The mean peak amplitude (microV) recorded over the left and right hemisphere under the two levels of workload during the neutral and depressed conditions.

Furthermore the interaction between **mood**, **workload**, **hemisphere** and **regions of interest** was significant ( $F(3,21) = 5.589$ ,  $p = 0.03$  (GG corrected) (Figure 7.10). It appears that the basis of this interaction lies within the frontal region of interest. During the neutral mood condition, there is little difference between left and right activation when participants perform the low workload level working memory task. As well as an increase in the peak negative amplitude recorded during the 3-back version of the task, the increased workload is associated with a larger difference in inter-hemispheric activity. During the depressed mood condition, there is greater negative activity associated with the 0-back condition in comparison with performance of this workload level under a neutral mood however a similar pattern of inter-hemispheric activity is seen. However in the 3-back version of the task, the inter-hemispheric pattern is reversed in comparison to the performance of this workload level under the neutral condition. Finally, it can be seen that similar levels of activation are recorded under the neutral 3-back and depressed 0-back conditions. The estimated groups means and standard errors calculated for this interaction are presented in Table 7.17.



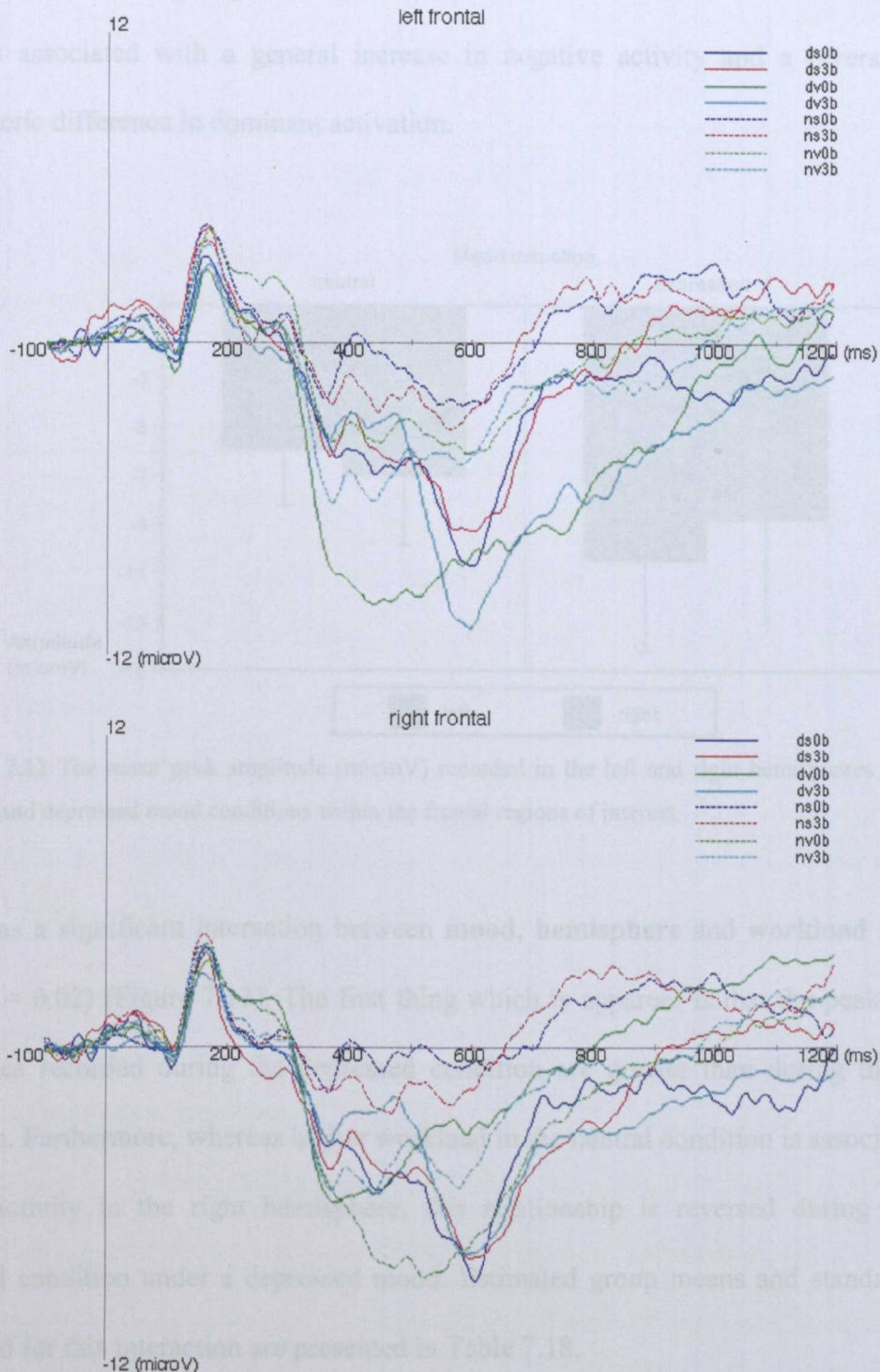


**Figure 7.10** The mean peak amplitude (microV) recorded over the left and right hemisphere under the two levels of workload within each region of interest during the neutral (A) and depressed (B) conditions.

			Frontal		Central		Parietal		Occipital	
			Mean (N = 8)	SE	Mean (N = 8)	SE	Mean (N = 8)	SE	Mean (N = 8)	SE
Neutral	0-back	Left	-4.21	2.04	0.79	0.49	0.45	0.63	-3.12	1.61
		Right	-4.17	1.64	1.22	0.65	-0.39	0.67	-5.03	1.43
	3-back	Left	-7.53	3.44	1.04	0.48	1.01	1.08	-2.66	1.50
		Right	-9.79	4.53	0.72	0.52	0.36	1.11	-3.76	1.31
Depressed	0-back	Left	-8.60	3.53	0.75	0.50	0.83	0.77	-2.35	1.05
		Right	-9.02	3.83	1.20	0.69	0.45	0.98	-3.44	1.26
	3-back	Left	-12.22	4.63	-0.46	0.99	1.46	1.47	-1.71	1.87
		Right	-8.39	4.98	0.59	0.79	0.13	1.28	-2.49	1.52

**Table 7.17** Mean peak amplitude (microV) and standard errors (SE) calculated for the during the 0-back and 3-back workload levels over the left and right hemisphere under the neutral and depressed conditions, within each region of interest.

As a number of interactions are associated with the frontal region of interest (Figure 7.11), a post hoc repeated measures ANOVA will be performed upon activity within this region.



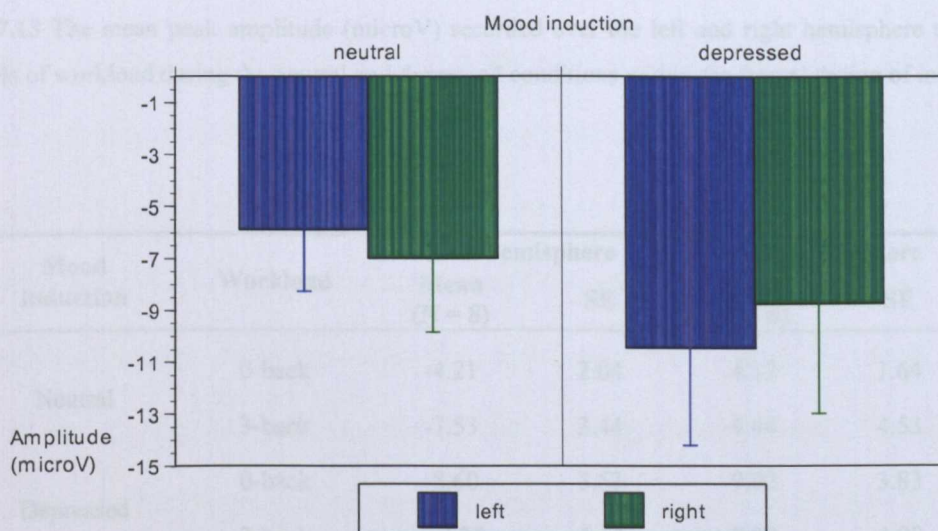
**Figure 7.11** The event-related potentials for correct response to verbal and spatial stimuli at 0-back and 3-back levels of workload under neutral and depressed mood conditions within the frontal region of interest.



#### 7.3.3.4.1 Frontal ROI post hoc analysis

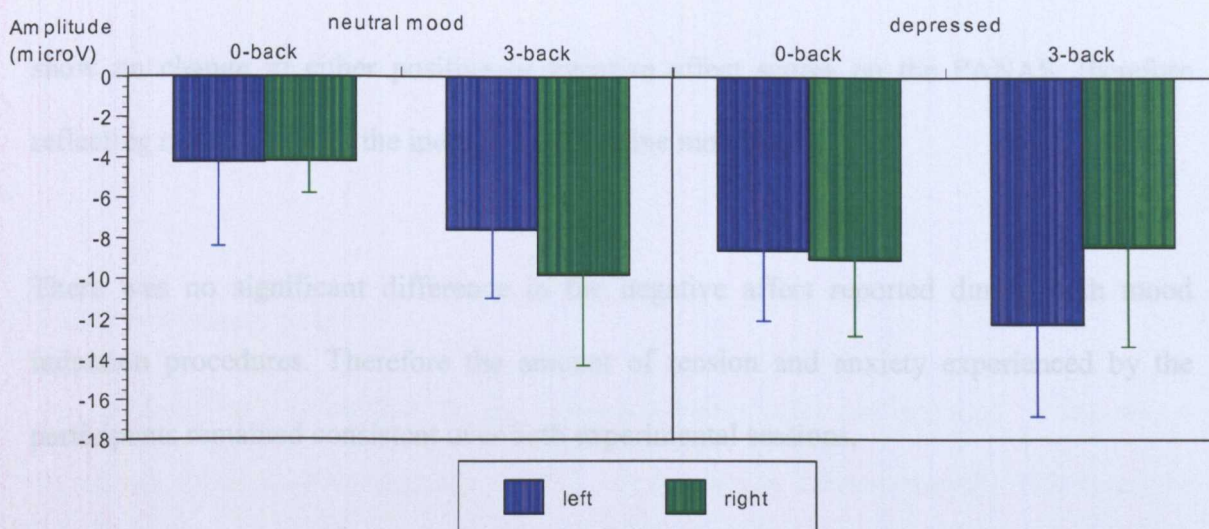
##### 7.3.3.4.1.1 Mood

The interaction between mood and hemisphere, although not significant ( $F(1,7) = 4.481$ ,  $p = 0.07$ ), is worth noting (Figure 7.12). In comparison with the neutral mood, the depressed mood is associated with a general increase in negative activity and a reversal in the hemispheric difference in dominant activation.



**Figure 7.12** The mean peak amplitude (microV) recorded in the left and right hemispheres under the neutral and depressed mood conditions within the frontal regions of interest.

There was a significant interaction between **mood**, **hemisphere** and **workload** ( $F(1,7) = 7.521$ ,  $p = 0.02$ ) (Figure 7.13). The first thing which is apparent is that the peak negative amplitudes recorded during the depressed condition are greater than during the neutral condition. Furthermore, whereas higher workload in the neutral condition is associated with greater activity in the right hemisphere, this relationship is reversed during the high workload condition under a depressed mood. Estimated group means and standard errors calculated for this interaction are presented in Table 7.18.



**Figure 7.13** The mean peak amplitude (microV) recorded over the left and right hemisphere under the two levels of workload during the neutral and depressed conditions within the frontal region of interest.

Mood induction	Workload	Left hemisphere		Right hemisphere	
		Mean (N = 8)	SE	Mean (N = 8)	SE
Neutral	0-back	-4.21	2.04	-4.17	1.64
	3-back	-7.53	3.44	-9.44	4.53
Depressed	0-back	-8.60	3.53	-9.02	3.83
	3-back	-12.22	4.63	-8.39	4.98

**Table 7.18** Mean peak amplitude (microV) and standard errors (SE) calculated for the left and right hemisphere for the 2 levels of workload under the neutral and depressed conditions, within the frontal region of interest.

## 7.4 Discussion

### 7.4.1 Mood induction procedure

The mood profile predicted during a depressed mood induction was that there would be no change in negative affect scores and a decrease in positive affects scores of the PANAS. This is representative of an increase in reported level of sadness, without it being associated with tension or anxiety. The mood profile associated with a neutral mood induction should



show no change in either positive or negative affect scores on the PANAS, therefore reflecting maintenance of the individual's baseline mood state.

There was no significant difference in the negative affect reported during both mood induction procedures. Therefore the amount of tension and anxiety experienced by the participants remained consistent over both experimental sessions.

There was no significant overall difference in the positive affect scores associated with the two mood induction procedures, but there was a significant main effect of test time ( $p = < 0.001$ ), which reflected an increase in the sadness experienced by participants over the experimental session. The pairwise comparison of this effect revealed significant increases in the degree of sadness experienced by the participants both during the mood induction procedures and during the performance of the working memory task. The interaction between mood induction procedure and test time neared significance ( $p = 0.06$ ). Both induction procedures were associated with a decrease in positive affect scores, however a larger decrease was associated with the depressed induction procedure. Therefore the mood profile is nearer to that expected for positive affect, with a greater increase in sadness associated with the depressed treatment than with the neutral treatment.

One possibility which arises from the significant main effect of test time would be to change the boundaries of the neutral and depressed mood condition. The sadness experienced by the participants increased over the performance of the working memory task, regardless of which induction procedure they had completed. Therefore, a distinction could be made between the first and second halves of the experiment to create the 'neutral'

and 'depressed' conditions. Unfortunately, reanalysis of the data in this way was not practical due to the reduction in data that would occur.

#### **7.4.2 Behavioural data**

As predicted task manipulations did influence behavioural performance. There was a significant effect of workload ( $p = 0.01$ ), where reaction times during the high workload 3-back task were slower than those recorded during the 0-back task. This indicates that the participants were experiencing an increase in cognitive workload when the number of items to be held and manipulated within working memory was increased to 3. There was also a main effect of modality ( $p = 0.05$ ), where the reaction times associated with the verbal task were slower than those recorded during the spatial task. Therefore the participants found the maintenance and manipulation of verbal information more difficult during the verbal task than the spatial task.

As a result of the cognitive workload increase associated with the 0-back and 3-back version of the working memory tasks, it was predicted that both the percentage error rate and reaction times would be sensitive to the mood manipulation. There were no significant effects associated with the percentage error rate, therefore the same level of performance was associated with task, regardless of task modality, workload levels and the mood at the time of performance. Although the mean reaction time in the depressed condition was slower than during the neutral condition, this difference was not significant. Therefore the second hypothesis is not supported as neither accuracy or speed deficits were associated with the depressed mood.

### 7.4.3 Electrophysiological data

Before the influence of mood can be assessed, the electrophysiological signature of the n-back task must be considered. The main effect of workload neared significance ( $p = 0.06$ ), which reflected a larger negative amplitude being associated with the 3-back condition of the working memory task. An increase in negative activity is thought to reflect increases in cognitive workload (Ruchkin et al., 1992), the trend recorded in this study supports this interpretation, and tentatively provides a means of indexing the influence of workload within this study.

There was a significant interaction between workload and time bin ( $p = 0.02$ ), where the difference in negativity associated with the 0-back and 3-back tasks was greatest during the earlier 300-500ms time bin. There are later components within event-related components recorded during working memory tasks, however these have been associated with task completion (Pelosi et al., 2000) rather than the active processes required during the performance of the working memory task. Therefore the maximal differentiation between the cognitive workload levels, as indexed by greater negative activation, occurs within 300-500ms post-stimulus presentation.

The topographical localisation of the hypothesis to the frontal region of interest was supported by the significant main effect of region of interest ( $p = 0.007$ ). The amplitude of peak activation varies across the scalp, however the greatest level of negative activation occurs within the frontal region. As this reflects a large degree of cognitive effort, this is consistent with the proposal that the active manipulation of information held within working memory tasks occurs in the prefrontal cortex (Ruchkin et al., 1992). In addition,

the magnitude of activation with this region is indicative of high levels of cognitive effort being associated with the task.

There was however, no lateralisation of activation as a result of the task modality. This result is more consistent with the haemodynamic neuroimaging studies which have shown bilateral activation during verbal and spatial tasks (Nystrom et al., 2000; Postle et al., 2000).

It was hypothesised negative activity within the frontal region of interest within the 300-500ms post-stimulus time been would be increased during the performance of a working memory task under a depressed mood. Furthermore, there would be a differential effect of increasing workload under the neutral and depressed moods. The Resource Allocation Model (Ellis et al., 1988) predicts that depression will be associated with performance deficits when the experience of the emotions leads to there being insufficient resources available to perform cognitive task. Furthermore, in circumstances where the experience of depression and the control of cognitive performance share a common resource reserve, the susceptibility to resource allocation problems will be heightened. As working memory has a high level of cognitive intensity and the active maintenance of information within working memory and the experience of depression are associated with activation of the dorsolateral prefrontal cortex, it was proposed that resource conflict would arise.

While the prediction that a depressed mood would alter frontal activity was not categorically supported, there is evidence that sadness modulates cortical activity within the

frontal lobes. Although not significant, it is worth noting the interaction between mood and hemisphere ( $p = 0.08$ ). The asymmetry recorded during the performance of the working memory task under a neutral mood was diminished during performance under a depressed mood. Increased levels of negativity during the performance of working memory tasks by depressed individuals have been previously reported (Pelosi et al., 2000), with a suggestion that it reflected the recruitment of additional neuronal populations. It is tentatively suggested that performance is lateralised to the right hemisphere under a neutral mood and the elimination of this asymmetry reflects neuronal compensatory activation.

This interpretation is supported by the significant interaction between mood, hemisphere and region of interest ( $p = 0.03$ ). As can be seen in Figure 7.8, there is a large degree of negative activation within the frontal region of interest, in comparison to the other regions across the scalp, and this anterior hemispheric asymmetry is modulated by the depressed mood. Under a neutral mood, the amplitude of negative activation is greater within the right hemisphere. The induction of a depressed mood is associated with an increase in left hemisphere activation, the magnitude of which results in the reversal of asymmetry. The localisation of this interaction to the frontal region of interest supports the postulation that the degree of interference will be maximal when the cognitive task and experience of emotion share the same limited resource capacity. Furthermore, the reversal of asymmetry during the depressed condition could be seen to reflect the compensatory recruitment of additional neuronal populations on the left hemisphere that are normally involved the control of the working memory task. The success of this is reflected in the maintenance of performance in the depressed mood condition.

Similarly, the interaction between mood, hemisphere and workload neared significance ( $p = 0.08$ ), which also reflected the reduction of hemispheric asymmetry during the depressed condition. It should be noted in Figure 7.9 that there is a similarity between the levels of activation associated with the performance of the 3-back working memory task under the neutral mood and the performance of the 0-back condition under the depressed mood.

This potential effect was elucidated by the significant interaction between mood, workload, hemisphere and region of interest (Fig. 7.10). The localisation of the interference effect to the frontal region of interest is again supported by the greater degree of anterior negative activation. The varying patterns of activation during the different levels of workload should also be noted. When participants perform the low cognitive workload task under a neutral mood, there is little difference in the activation of the frontal region, with both hemispheres showing similar levels of negative activation. As the workload is increased from the 0- to the 3-back version of the task, the amplitude of negative activity increases within the frontal region and the asymmetry between the left and right hemisphere becomes apparent. Furthermore, similar levels of negativity were associated with the performance of the 3-back task under a neutral mood and the depressed 0-back condition. Additionally, as the cognitive workload is increased during the 3-back version of the task, a further increase in the magnitude of negative activity occurs which leads to a reversal of asymmetry to dominance within the left hemisphere.

It could be argued that as the PANAS results do not support the presence of an induced mood, the increases in negative activity reported reflect the novelty of the 3-back task in comparison with the 0-back version. However, as the participants were able to practice the

3-back version of the tasks it is unlikely that increase in difficulty would be a surprise to them. Furthermore, the difference in cortical activity between the 0-back and 3-back tasks is not mirrored across the two 'mood' conditions. As the use of the neutral and depressed induction procedures was counterbalanced across participants, it would seem that the differential activation was related to the mood state of the participant. However, this conclusion must remain tentative due to the verbal differentiation between the mood states only.

Consequently, it appears that the magnitude of negative activity is a sensitive index of cognitive workload. In general, the increase in workload associated with the 0-back and 3-back task is reflected as an increase in negative activity within the frontal region of interest. Within the neutral mood condition, it can be seen that the performance of the 3-back working memory task is associated with an increase in negativity and the development of asymmetrical hemispheric activation. The change in mood could in itself be interpreted as an increase in workload as the performance of the 0-back task under the depressed mood is associated with greater negativity than during the neutral condition.

Furthermore, the performance of this low cognitive workload task under the depressed mood is associated with the same degree of cortical activation as performance of the high cognitive workload task under the neutral mood. In addition there is evidence of a requirement of additional cognitive effort as the hemispheric asymmetry is reduced. When the level of cognitive workload is increased within the depressed condition, the increase in activity within the left hemisphere is such that the asymmetry present in the performance of

this task in the neutral mood is reversed. This infers that there is compensatory activation of additional neuronal populations required to perform this task in the depressed condition.

Through the performance of a post hoc analysis upon the frontal region of interest alone, the cortical activation associated with the performance of a working memory task under a neutral and depressed mood was investigated further. As hypothesised there were higher levels of negativity on the left hemisphere in the depressed mood which neared significance ( $p=0.07$ ), providing some support for the findings of Pelosi et al. (2000) and Deldin et al. (2001). However the significant interaction between mood, hemisphere and workload ( $p = 0.02$ ), provided a more substantial support of this finding.

The performance of the working memory task under a depressed mood can be viewed as creating an increase in cognitive workload. When the performance of the 0-back condition is compared under neutral and depressed moods it can be seen that the depressed mood is associated with an increase in negative activity. As there was no interaction between mood and workload in terms of reaction times, it can be seen that this increase in cortical activity maintained the behaviour performance associated with the 0-back task.

Under both the conditions, the increase in cognitive workload which occurred when the task demands were raised from a 0-back to 3-back task was associated with an increase in negativity. When the 3-back task was performed under the neutral mood, there was an asymmetry of activation reflected as increased negativity within the right hemisphere. However, when the same task was performed under the depressed condition there was a



reversal in asymmetry, where the increase in negative activation within the left hemisphere led to it becoming associated with a greater degree of negative activation. This could be interpreted as indicating the existence of compensatory activation by neurons not normally associated with the performance of a 3-back working memory task.

In summary, this study cautiously indicates that there are changes in cortical activity associated with increasing working memory demands during transient mood changes, which are not evident at the subjective and behavioural levels. In accordance with the Resource Allocation model, changes were more apparent in a task which was associated with increased cognitive intensity and a common neuroanatomical basis. The location of these changes mainly within the left frontal areas is in accordance with neuroanatomical models of working memory and of affective control.

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## Chapter 8

### General conclusion

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#### 8.1 The influence of mood on cognitive performance

The Resource Allocation Model (Ellis et al., 1988) provides a fully integrated account of the influence of affect on cognitive performance. It predicts that emotional states modulate the amount of capacity that is allocated to a criterion task through the elicitation of mood congruent memories (Ellis et al., 1988). The effect of these memories will be dependent upon the relationship between the information contained within them and that associated with the criterion task (Ellis et al., 1988; Ellis et al., 1993). Therefore task-relevant thoughts will lead to performance facilitation (for example, Isen et al., 1987) and task-irrelevant thoughts will lead to performance suppression (for example, Oaksford et al., 1996).

Through theoretical and behavioural explorations, it has been hypothesised that the location for the affect-cognition interaction is the central executive component of working memory (Oaksford et al., 1996), the neurological correlates of which lie within the dorsolateral region of the prefrontal cortex (see for example, Petrides, 2000a). As well as being fundamental to the control of cognitive processing, this region of the brain has also been shown to play an important role in the experience of depressive episodes (Dolan et al., 1993) and induced depressed mood states (Drevets, 2000).

It is postulated in situations where the experience of emotion and control of cognitive processes share a common neuroanatomical basis, there will be increased sensitivity to the affect-cognition interaction proposed by the Resource Allocation Model. Through the manipulation of cognitive complexity in tasks associated with the activation of the frontal lobes, this thesis aimed to investigate the mood modulation of the affect-cognition interaction at a behavioural and cortical level.

## **8.2 An electrophysiological approach**

The millisecond temporal resolution possible with ERP data offers the possibility of tracking the time course of different components of complex cognitive processes. Changes in the amplitude of specific components can be interpreted as indices of the allocation of specific cortical resources to the task, with increases associated with stronger electrical fields, and decreased activation or reduction in amplitude associated with a deficit in the allocation of resources (Kayser et al., 2001). It is also possible that increasing amplitude is associated with wider recruitment of underlying cortical areas (Kayser et al., 2001). In general, comparisons can be made between the structural and topographical characteristics of specific components in different conditions to investigate the effects of the independent variables of interest.

## **8.3 Mood induction procedures**

As the aim of this thesis was to investigate the affect-cognition interaction, a mood induction procedure was required that was able to induce reflective emotions. Rather than being a transient reaction to emotional stimuli, reflective emotions are complex experiences which have cognitive, somatic and emotional components. Following an assessment of mood induction techniques, it was concluded that the False Feedback Procedure,

Autobiographical Recall and the Velten mood induction procedures were all capable of inducing these kinds of emotions.

A comparative study (Chapter 4) indicated that the mood profiles associated with a depressed state were more consistently achieved than those associated with elated state. The mood profile expected during a depressed mood state predicted no change in negative affect scores and a decrease in positive affect as measured by the sub scales of the PANAS (Watson et al., 1988). This reflects the maintenance of negative affect state, where there is no increase in anxiety or tension, and a decrease in positive affect state, which represents an increase in sadness. Alternatively, a neutral mood induction should show no change in either negative or positive affect scores on the PANAS, thereby reflecting maintenance of the individual's baseline mood state.

The Velten mood induction procedure was chosen as it produced the appropriate mood profile and a depressed mood which was significantly different from the neutral state. However, in the studies reported here, the mood induction procedures were only associated with trends in the appropriate direction rather than significant changes in mood profile scores. One possibility is that the PANAS is insufficiently sensitive to the mild mood changes associated with mood induction. Alternatively, it is possible that participants rationalise their emotions during the mood assessment as they are aware of the transient nature of their experience. Consequently, the conclusions of this thesis must be assessed in light of the limited effectiveness of the mood induction procedure.

#### **8.4 Mood and passive attention**

Dense-array electrophysiological recordings were made while participants performed an oddball task under a neutral and depressed mood (Chapter 5). In terms of cognitive complexity, the processes thought to underlie the P300 component can be seen as representing low level information processing. Furthermore, the neuronal generators of the component (Smith et al., 1990) do not share a common neuroanatomical basis with those involved with the experience of emotion. Therefore it is assumed that there will be sufficient resources available to efficiently perform the oddball task whilst the participant is experiencing an induced depressed mood and there would be no change parietal cortical activation. As the neuroanatomical correlates of depression lie within the prefrontal cortex (Dolan et al., 1993), any electrophysiological manifestations of the induced mood were predicted to occur within the frontal region of interest.

The results indicated there being a potential blurring of the stimulus and hemispheric differences associated with task performance under a depressed mood. There were no differences with the reaction times associated with this particular task, however it is possible that the maintenance of behavioural performance is through the compensatory recruitment of additional neuronal populations. A reduction in asymmetry was reported which was associated with an increase in activation, on the left hemisphere. Therefore, even during a low intensity passive attention task, transient changes in mood produced changes in cortical activity that were not evident at the subjective or behavioural level.

#### **8.5 Mood and selective attention**

Dense-array electrophysiological recordings were made while participants performed a Stroop task under a neutral and depressed mood (Chapter 6). In terms of cognitive

complexity, the processes thought to underlie the Stroop task can be seen as representing moderate level information processing. The neuronal generators of context processing share a common neuroanatomical basis with those involved with the experience of emotion (George et al., 1994), however semantic processing of incongruent stimuli has been linked to temporo-parietal regions. Therefore it is possible the distribution of cortical activation will counteract the effects of insufficient resources available to efficiently perform the Stroop task whilst the participant is experiencing an induced depressed mood.

The Stroop effect was effectively demonstrated at the behavioural and partly at the cortical level with incongruent stimuli associated with greater negative activation in central regions. At the subjective and behavioural level, the mood manipulation was not effective, but at the cortical level there was evidence of difference in activation between the two mood conditions. As in the previous study, this difference is associated with a reduction in asymmetrical activation in the depressed condition, in this case associated with asymmetries on the right hemisphere. Such reductions in asymmetry could be associated with an increase in activation to produce the same level of task performance, a form of compensatory recruitment.

### **8.6 Mood and sustained attention**

Dense-array electrophysiological recordings were made while participants performed a working memory task under a neutral and depressed mood (Chapter 7). In terms of cognitive complexity, working memory represents a high level information processing. As the neuronal generators of working memory share a common neuroanatomical basis with those involved with the experience of emotion, it is assumed that there will be insufficient

resources available to efficiently perform a working memory task whilst the participant is experiencing an induced depressed mood.

Negative activity proved to be a sensitive index of cognitive work load as it was able to differentiate between f 0-back and 3-back working memory tasks under neutral and depressed moods. A clear distinction between workload intensity was found within each mood condition, furthermore it appears that the experience of a depressed mood is analogous to increased cognitive workload. Similar neurological signatures were associated with the performance of a high workload 3-back working memory task under a neutral mood and the performance of a low workload 0-back working memory task under a depressed mood. The high level cognitive workload was associated with a reversal of asymmetry under the two mood conditions, with performance under a depressed mood being associated with an increase in negativity which led to shift in dominant activation. Therefore creating a anterior asymmetry consistent with Davidson's (1998) model of emotional activation within the prefrontal cortex.

### **8.7 Affect-Cognition Interaction**

Consistently, the transient effects of induced mood brought about changes in cortical activity that was not apparent when subjective and behavioural indices of performance were employed. The results of this thesis provide support that the location of the affect-cognition interaction described by the Resource Allocation Model (Ellis et al., 1988) are located within anterior regions. Furthermore, increased cognitive complexity and a shared reliance upon the limited resources associated with the prefrontal cortex were related to the likelihood that an affect-cognition interaction would occur.

Working memory, which is both high in cognitive complexity and shares its neurological correlates with depression, was most susceptible to the affection-cognition interaction. Using negative activity as an electrophysiological index of cognitive workload, increase in negative amplitude were recorded over the frontal regions of interest during the performance of a working memory task under a depressed condition. Furthermore, the performance of the high workload 3-back task was associated with an increase in left hemisphere activation to such an extent that hemispheric asymmetries were reversed.

It is tentatively concluded that the experience of a transient mood causes a sufficient burden upon limited resources within the prefrontal cortex, that there are insufficient resources available for the performance of complex cognitive tasks also controlled by this region. Increases in negative activity suggest that compensatory activation of neuronal populations not normally recruited in the performance of such tasks ensures satisfactory performance.

### **8.8 Future directions**

There are four main directions in which the findings of this thesis should be taken. Firstly, it is apparent that cortical activity is more sensitive than a subjective mood assessment to the intensity of mood associated with the Velten induction procedure. As an electroencephalographic recording was made during the induction period, it would be interesting to see if EEG activity was specific to the neutral and depressed mood states. This would allow the apparent insensitivity of objective measure of mood to be supplemented with an objective index of mood.

Dense array EEG lend themselves to the application of source localisation algorithms, although this technique was not utilised in this thesis. As the modulation of the affect-



cognition interaction was localised to the frontal regions of interest, it would be interesting to expand the analysis to include the application of source localisation routines. This would allow the neuronal generators of the distribution of electrical activity across the scalp to be determined, and therefore allow the role of the dorsolateral prefrontal cortex within the affect-cognition interaction to be specifically investigated.

The results of working memory study point to the cognitive intensity as being the task characteristic most sensitive to affect-cognition interaction. It would be interesting to see if other tasks which required sustained attention show similar cortical differences. There are a number of tasks which would afford themselves to this kind of investigation, for example the Bakan task (Bakan, 1959). In this vigilance task, participants are presented with a random series of number which vary from 1 to 9, and their task is to respond when either three odd numbers or three even numbers have been consecutively presented. Not only is the task sufficiently intense, but the presentation characteristics lend themselves to the formation of event-related potentials.

Finally, it is apparent that the intensity of the mood produced by the Velten mood induction procedure was not very high. Evidence presented in this thesis tentatively suggests the modulation of frontal activity by a sad mood, but none of the experiments provided unequivocal support of this hypothesis. One possible way of increasing the affective experience of the participant would be to use affective stimuli, therefore maintaining the affective state throughout the experimental session. An emotional Stroop is already used within the research of emotional perception (Richards, French, Johnson, Naparstek, & Williams, 1992), and both the oddball paradigm and the n-back working memory task lend

themselves to the use of affective stimuli. By creating a 'mood context' for the participant, they would be less likely to rationalise away the changes in mood they are experiencing.

## **8.9 Conclusions**

Through the use of dense-array electrophysiological techniques, this thesis has investigated the affect-cognitive interaction. An increase in negative activity recorded over the prefrontal cortex implies that increased cortical activation is required to ensure effective performance of cognitively intense tasks during the experience of a transient mood state. As the Resource Allocation Model (Ellis et al. 1988) postulates that a depressed mood leads to a reduction in the capacity of resources allocated to the control of cognitive tasks, the increase in negative activity suggests that compensatory activation of neuronal populations, not normally recruited in the performance of such tasks, ensures satisfactory performance.

# Appendix 1

## Mood Induction Techniques

Citation	Investigation	Induction technique	Mood assessment	Manipulation check	Mood removal
Albersnagel (1988)	Induction technique	Velten	Visual Analogue Mood Scales (VAS; McCormack, Horne & Sheather 1988)	Pre and post induction	None detailed
Albersnagel (1988)	Induction technique	Music	VAS	Pre and post induction	None detailed
Alexander & Guenther (1986)	Mood and memory	Velten	None	None	E-VMIP statements
Alloy, Abramson & Viscusi (1981)	Mood and judgements of control	Velten	Multiple Affect Adjective Checklist (MAACL; Zuckerman & Lubin 1965)	Pre and post induction	Baseline mood re-achieved – experimenter convinced
Baker & Guttfreund (1993)	Induction technique	Autobiographical recall	Depression Adjective Checklist (DACL; Lubin 1965); State Anxiety Inventory (STAI; Spielberger, Gorsuch & Lushene 1968)	Pre and post induction	Debrief - no details
Bartolic, Basso, Schefft, Glauser & Titanic-Schefft (1999)	Mood and cognition	Velten	MAACL	Pre and post induction	Debrief - no details
Baucom & Aiken (1981)	Mood and eating	False feedback	DACL	Post induction	Debrief - no details
Beck & McBee (1995)	Mood and memory	Velten (altered)	Positive And Negative Affect Scale (PANAS; Watson, Clark & Tellegen 1988)	Pre and post induction	None detailed

Citation	Investigation	Induction technique	Mood assessment	Manipulation check	Mood removal
Berkowitz (1987)	Mood and willingness to help	Velten	Study specific	Post induction	None detailed
Berkowitz (1987)	Mood and willingness to help	Autobiographical recall	Study specific	Post induction	None detailed
Bodenhausen, Gabriel & Lineberger (2000)	Mood and cognition	Autobiographical recall	Study specific	Post induction	Humorous cartoons
Boiten (1996)	Methodological detail	Facial manipulation	Study specific	Post induction	None detailed
Bollenbach & Madigan (1982)	Mood and cognition	Velten	Study specific	Post induction	E-VMIP statements
Bradley, Cuthbert & Lang (1996)	Mood and sensitisation	International Affective Picture System (IAPS; Lang et al 1988)	Startle probe	Pre and post induction	None detailed
Bradley, Greenwald, Petry & Lang (1992)	Mood and memory	IAPS	Self Assessment Manikins (SAM; Lang 1980)	Post induction	None detailed
Brewer, Doughtie & Lubin (1980)	Induction technique	Autobiographical recall	DACL; STAI; Beck Depression Inventory (BDI; Beck et al 1961)	Pre and post induction	Debrief and discussion
Brewer, Doughtie & Lubin (1980)	Induction technique	Velten	DACL; STAI; BDI	Pre and post induction	Debrief and discussion
Brewin & Harris (1985)	Mood and attribution	Music	BDI	Pre and post induction	Debrief - experimenter convinced
Brown & Taylor (1986)	Mood and memory	Velten	Study specific	Post induction	E-VMIP statements
Brown & Taylor (1986)	Mood and memory	Autobiographical recall	Study specific	Post induction	None detailed
Buchwald, Strack & Coyne (1981)	Induction techniques	Velten	MAACL	Post induction	None detailed
Cairns & Norton (1988)	Induction technique	Velten	MAACL	Pre and post induction	E-VMIP statements
Carson & Adams (1980)	Mood and activity	Velten	DACL	Pre and post induction	None detailed
Casanova, Katkovsky & Hershberger (1988)	Theories of depression	False feedback	STAI; The Paranoid Adjective Checklist (PAC; Katkovsky 1986)	Pre and post induction	Debrief - no details

<b>Citation</b>	<b>Investigation</b>	<b>Induction technique</b>	<b>Mood assessment</b>	<b>Manipulation check</b>	<b>Mood removal</b>
Cash, Rimm & MacKinnon (1986)	Induction technique	Velten	MAACL	Pre and post induction	Positive imagery/relaxation tape and an interview
Chartier & Ranieri (1989)	Induction technique	Velten	DACL	Pre and post induction	Debrief - no details
Chartier & Ranieri (1989)	Induction technique	False feedback	DACL	Pre and post induction	Debrief - no details
Clark & Teasdale (1985)	Mood and memory	Music	Study specific	Pre and post induction	Baseline mood re-achieved (no details)
Cohen, Towbes & Flocco (1988)	Mood and perception of life events	Velten	MAACL	Post induction	E-VMIP statements
Crucian, Hughes, Barrett, Williamson, Bauer, Bowers & Heilman (2000)	Emotion and visceral feedback	Affective pictures (study specific)	Attractiveness rating of stimuli and heart rate	Post induction (although screened for depression using the Self-Rating Depression Scale (Zung 1965))	None detailed
Cunningham, Schaffer, Barbee, Wolff & Kelley (1990)	Mood and willingness to help	Velten	Study specific	Post induction	E-VMIP statements
Cuthbert, Bradley & Lang (1996)	Mood and activation	IAPS	SAM and startle	Post induction	None detailed
Danzer, Dale & Klions (1990)	Theories of depression	Velten	MAACL	Pre and post induction	E-VMIP statements
Davidson, Ekman, Saron, Senulis & Friesen (1990)	Neurological correlates of emotion	Film	Study specific	Post induction	None detailed
Delp & Sackheim (1987)	Measures of mood	Autobiographical recall	MAACL	Pre and post induction	None detailed
Duncan & Laird (1980)	Mood and self perception	Facial manipulation	Nowlis Mood Adjective Checklist (Nowlis MACL; Nowlis 1965)	Post induction	None detailed
Dykman (1997)	Theories of depression	Velten and music	MAACL	Pre and post induction	E-VMIP statements
Egloff (1998)	Emotion	False feedback	PANAS	Pre and post induction	Debrief -no details

Citation	Investigation	Induction technique	Mood assessment	Manipulation check	Mood removal
Ekman, Friesen & Ancoli (1980)	Mood and facial action	Film	Study specific	Pre and post induction	None detailed
Ellis, Ottaway, Varner, Becker & Moore (1997)	Mood and comprehension	Velten (altered)	DACL	Post induction	E-VMIP statements
Ellis, Thomas, McFarland & Lane (1985)	Mood and memory	Velten	DACL	Post induction	E-VMIP statements
Ellis, Thomas, & Rodriguez (1984)	Mood and memory	Velten	DACL	Post induction	E-VMIP statements
Fiedler, Pampe & Scherf (1986)	Mood and memory	Velten (translated)	Study specific	Post induction	None detailed
Fiedler & Strohm (1986)	Mood and memory	Autobiographical recall	None	None	None detailed
Foa, McNally & Murdock (1989)	Mood and memory	Natural (public speaking)	VAS; SAM; STAI	Pre and post induction	None detailed
Forgas (1999)	Mood and requests	Autobiographical recall	Study specific	Post induction	Debrief - no details
Forgas (1998)	Mood and attribution	False feedback	Study specific	Post induction	Baseline mood re-achieved (no details)
Forgas (1990)	Mood and group judgements	Film	Study specific	Post induction	None detailed
Forgas, Bower & Moylan (1990)	Mood and self perception	False feedback	Study specific	Post induction	Baseline mood re-achieved (no details)
Forgas, Bower & Moylan (1990)	Mood and self perception	Film	Study specific	Post induction	Debrief only
Fox, Knight & Zelinski (1998)	Induction technique	Velten and music	DACL	Post induction	"Happy" music
Frost & Green (1982)	Induction techniques	Velten	Personal Feeling Scale (PFS; Frost et al 1979); BDI; MAACL	Pre and post induction	E-VMIP statements
Frost, Graf & Becker (1979)	Theories of depression	Velten	MAACL; study specific	Pre and post induction	E-VMIP statements
Gage & Safer (1985)	Mood and emotional face recognition	Imagination	Study specific	Post induction	Baseline mood re-achieved (no detail)

<b>Citation</b>	<b>Investigation</b>	<b>Induction technique</b>	<b>Mood assessment</b>	<b>Manipulation check</b>	<b>Mood removal</b>
Gilboa-Schechtman, Revelle & Gotlib (2000)	Mood and cognition	Autobiographical recall and music	VAS	Post induction	Recollection of neutral experience (baseline mood recorded now)
Goldstein & Strube (1994)	Independence of emotions	Natural (exams)	PANAS	Pre and post induction	None detailed
Gomez, Cooper & Gomez (2000)	Susceptibility to induction	False feedback	PANAS	Pre and post induction	None detailed
Gotlib & McCann (1984)	Theories of depression	Velten	MAACL	Post induction	E-VMIP statements
Gross & Levenson (1995)	Induction technique	Film	Modified Differential Emotions Scale (MDES; Gross & Levenson 1995)	Post induction	None detailed
Hale & Strickland (1976)	Mood and cognition	Velten	Study specific	Post induction	None detailed
Hänze & Meyer (1998)	Mood and priming	Film	Study specific	Post induction	None detailed
Hawkins, French, Crawford & Enzle (1988)	Mood and time perception	Velten	Study specific	Post induction	E-VMIP statements
Henderson & Lohr (1982)	Theories of depression	Velten	MAACL	Post induction	None detailed
Hockey, Maule, Clough & Bdzola (2000)	Mood and cognition	False feedback	Study specific	Pre and post induction	None detailed
Holdwick & Wingenfeld (1999)	Methodological detail	Autobiographical recall	MAACL-Revised (MAACL-R; Zuckerman & Lubin 1985)	Pre and post induction	None detailed
Ingram (1984)	Mood and feedback	False feedback	MAACL	Post induction	Debrief – no details
Isen & Daubman (1984)	Mood and categorisation	Film	Study specific	Post induction	None detailed
Isen & Daubman (1984)	Mood and categorization	Gift	Study specific	Post induction	None detailed
Isen & Gorgoglione (1983)	Induction technique	Velten	Study specific	Post induction	None detailed
Isen & Gorgoglione (1983)	Induction technique	Film	Study specific	Post induction	None detailed

Citation	Investigation	Induction technique	Mood assessment	Manipulation check	Mood removal
Isen, Daubman & Nowicki (1987)	Mood and problem solving	Film	Study specific	Post induction	Debrief - no details
Isen, Johnson, Mertz & Robinson (1985)	Mood and word associations	Film	Study specific	Post induction	None detailed
Isen, Shalker, Clark & Karp (1978)	Mood and memory	Gift	None	None	None detailed
Isen, Shalker, Clark & Karp (1978)	Mood and memory	False feedback	None	None	None detailed
Johnson & Tversky (1983)	Mood and the perception of risk	Story	Study specific	Post induction	None detailed
Kaufmann & Vosburg (1997)	Mood and problem solving	Film	Russell Adjective Checklist (Russell 1979)	Post induction	Debrief - no details
Kenealy (1997)	Mood and memory	Velten	Study specific	Post induction	None detailed
Kenealy (1997)	Mood and memory	Velten	Study specific	Post induction	None detailed
Kenealy (1988)	Induction technique	Music	VAS	Post induction	None detailed
Kulbartz-Klatt, Florin & Pook (1999)	Mood and self perception	Autobiographical recall and music	VAS	Pre and post induction	None detailed
Kumari, Hemsley, Cotter, Checkley & Gray (1998)	Mood and memory	Drug	UWIST Mood Adjective Checklist (UMACL; Matthews, Jones & Chamberlain 1990)	Pre and post induction	Natural and drug to remove possible side effects
Kuykendall, Keating & Wagaman (1988)	Methodological detail	Velten	MAACL; judging words	Post induction	None detailed
Kwaitkowski & Parkinson (1994)	Theories of depression	Velten	DACL	Pre and post induction	E-VMIP statements
Laird, Wagener, Halal & Szegda (1982)	Mood and memory	Facial manipulation	Study specific	Post induction	None detailed
Lane, Reiman, Ahern, Schwartz & Davidson (1997)	Neurological correlates of emotion	Film	VAS	Post induction	None detailed
Lane, Reiman, Bradley, Lang, Ahern, Davidson, Schwartz (1997)	Neurological correlates of emotion	IAPS	SAM; Positron Emission Tomographic (PET) measurements	Pre and post induction	None detailed



<b>Citation</b>	<b>Investigation</b>	<b>Induction technique</b>	<b>Mood assessment</b>	<b>Manipulation check</b>	<b>Mood removal</b>
Lenton & Martin (1991)	Induction technique	Music	VAS	Post induction	None detailed
Levenson, Ekman & Friesen (1990)	Mood and facial action	Facial action	Study specific	Post induction	None detailed
Lewis & Harder (1988)	Induction technique	Velten	MAACL	Pre and post induction	Debrief – no details
Madigan & Bollenbach (1986)	Mood and irrational belief	Velten	MAACL	Post induction	E-VMIP statements
Madigan & Bollenbach (1982)	Mood and memory	Velten	Study specific	Post induction	E-VMIP statements
Manucia, Banmann & Gialdini (1984)	Mood and willingness to help	Autobiographical recall	Study specific	Post induction	None detailed
Marston, Hart, Hileman & Faunce (1984)	Induction technique	Film	Study specific	Pre and post induction	None detailed
Master & Miller (1991)	Theories of depression	Velten (adapted)	MAACL	Pre and post induction	None detailed
Matthews & Bradley (1983)	Mood and memory	Velten (adapted) and music	DACL	Pre and post induction	None detailed
McArthur, Solomon, & Jaffe (1980)	Neuroanatomical correlates of emotion	Facial manipulations	Nowlis MAACL	Post induction	None detailed
McFarland & Buehler, (1997)	Mood and memory	False feedback	Study specific	Post induction	None detailed
McFarland & Ross (1982)	Mood and attribution	False feedback	Study specific	Post induction	None detailed
McHugh & Wierzbicki (1998)	Induction technique	Velten (adapted)	MAACL	Pre and post induction	E-VMIP statements
Melton (1995)	Mood and performance	Story	Study specific	Post induction	None detailed
Mills & Salkovskis (1988)	Mood and phobic reactions	Music	Study specific	Post induction	None detailed
Miranda & Persons (1988)	Mood and attitude	Velten	MAACL	Pre and post induction	None detailed

Citation	Investigation	Induction technique	Mood assessment	Manipulation check	Mood removal
Miranda, Gross, Persons & Hahn (1998)	Theories of depression	Film	Study specific	Pre and post induction	None detailed
Mitchell & Madigan (1984)	Mood and cognition	Velten	MAACL	Post induction	E-VMIP statements
Mukherji, Abramson & Martin (1982)	Mood and attribution	Velten	MAACL	Pre and post induction	Debrief - experimenter convinced
Müller, Keil, Gruber & Elbert (1999)	Neurological correlates of emotion	IAPS	SAM	Post induction	None detailed
Murray, Sujan, Hirt & Sujan (1990)	Mood and categorization	Velten	Study specific	Post induction	E-VMIP statements
Nagata & Trierweiler (1988)	Induction technique	Velten	MAACL	Post induction	E-VMIP statements
Oaksford, Morris, Grainger & Williams (1996)	Mood and cognition	Film	Study specific	Post induction	Debrief - no details
Orton, Beiman, LaPointe & Lankford (1983)	Methodological detail	Velten	DACL; STAI	Pre and post induction	None detailed
Pardo, Pardo & Raichle (1993)	Neurological correlates of emotion	Autobiographical recall	VAS; PET measurements	Post induction	None detailed
Parrott (1991)	Induction technique	Music	Study specific	Pre and post induction	Debrief - no details
Parrott & Sabini (1990)	Mood and categorization	Music	Study specific	Pre and post induction	None detailed
Pignatiello, Camp & Rasar (1986)	Induction technique	Music	DACL	Post induction	None detailed
Philippot (1993)	Induction technique	Film	Differential Emotions Scales (DES; Izard 1971); study specific	Post induction	Debrief - no details
Polivy & Doyle (1980)	Induction techniques	Velten	MAACL	Post induction	Debrief - no details
Radenhausen (1989)	Mood and self perception	Film	Study specific	Post induction	Debrief - experimenter convinced

<b>Citation</b>	<b>Investigation</b>	<b>Induction technique</b>	<b>Mood assessment</b>	<b>Manipulation check</b>	<b>Mood removal</b>
Ranieri & Zeiss (1984)	Theories of depression	Velten	DACL	Pre, middle and post induction	If final DACL is 4 points above baseline then recital of E-VMIP statements
Reiman, Lane, Ahern, Schwartz, Davidson, Friston, Yun & Chen (1997)	Neurological correlates of emotion	Film	VAS; PET measurements	Post induction	None detailed
Rexford & Wierzbicki (1989)	Induction technique	Velten (adapted)	MAACL	Pre and post induction	E-VMIP statements
Reynolds & Salkovskis (1992)	Mood and intrusive thoughts	Music	VAS	Pre and post induction	None detailed
Rholes, Riskind & Lane (1987)	Mood and memory	Velten	Study specific	Pre and post induction	None detailed
Richards, French, Johnson, Naparstek & Williams (1992)	Mood and performance	Pictures	SAI	Pre and post induction	None detailed
Riskind, Rholes & Eggers (1982)	Mood and memory	Velten	Study specific	Pre and post induction	None detailed
Ritz, Dahme & Claussen (1999)	Mood and muscle activity	Velten and IAPS	Study specific; VAS; SAM	Post induction	None detailed
Robins (1988)	Induction technique	Story	DACL	Pre and post induction	Positive story
Rodrigue, Olson & Markley (1987)	Mood and curiosity	Velten	MAACL	Pre and post induction	E-VMIP statements
Rogers, Miller, Mayer & Duval (1982)	Mood and willingness to help	Velten	MAACL	Post induction (Velten (1967) spontaneous associations used for pre-induction)	E-VMIP statements
Rosenhan, Salovey & Hargis (1981)	Mood and altruism	Story	Study specific	Post induction	None detailed
Russell & McAuley (1986)	Mood and success	Story	Study specific	Post induction	None detailed

<b>Citation</b>	<b>Investigation</b>	<b>Induction technique</b>	<b>Mood assessment</b>	<b>Manipulation check</b>	<b>Mood removal</b>
Salovey & Birnbaum (1989)	Mood and cognition	Autobiographical recall	Profile of Mood State (POMS; McNair 1970), DES; study specific	Pre induction Post induction	None detailed
Sawchuk, Lohr, Lee & Tolin (1999)	Mood and phobic reactions	Film	MDES	Post induction	None detailed
Schare & Lisman (1984)	Induction technique	Velten	MAACL	Pre and post induction	None detailed
Schare, Lisman & Spear (1984)	Mood and learning	Velten	MAACL	Post induction	None detailed
Schiff & Lamon (1994)	Induction technique	Muscle manipulation	Emotional tone of stories	Post induction	None detailed
Schotte, Cools & McNally (1990)	Mood and overeating	Film	POMS; VAS	Pre and post induction	Debrief - no details
Schwarz & Clore (1983)	Mood and perception of life events	Autobiographical recall	Study specific	Post induction	None detailed
Sherwood, Schroeder, Abrami & Alden (1981)	Induction technique	Velten	MAACL	Post induction	None detailed
Sirota & Schwartz (1982)	Mood and facial activity	Velten	HSAFAD Scale (Schwartz & Weinberger 1980)	Pre and post induction	None detailed
Sirota, Schwartz & Kristeller (1987)	Mood and facial activity	Velten	HSAFAD	Pre and post induction	E-VMIP statements
Slyker & McNally (1991)	Induction technique	Velten	VAS; MAACL	Pre and post induction	None detailed
Slyker & McNally (1991)	Induction technique	Music	VAS; MAACL	Pre and post induction	None detailed
Slyker & McNally (1991)	Induction technique	Velten and music	VAS; MAACL	Pre and post induction	None detailed
Small & Robins (1988)	Mood and recognition	Velten	None	None	None detailed
Snyder & White (1982)	Mood and memory	Velten	Study specific	Post induction	VMIP statements

Citation	Investigation	Induction technique	Mood assessment	Manipulation check	Mood removal
Sobotka, Davidson & Senulis (1992)	Neurological correlates of emotion	False feedback	Study specific	Post induction	None detailed
Stein, Goldman & Del Boca (2000)	Mood and alcohol consumption	Music	PANAS	Pre and post induction	None – positive mood only
Spence, Shapiro & Zaidel (1996)	Neurological correlates of emotion	IAPS	Study specific	Post induction	None detailed
Stemler (1982)	Anatomical correlates of emotion	Story	Study specific	Post induction	None detailed
Stemler (1982)	Anatomical correlates of emotion	Autobiographical recall	Study specific	Post induction	None detailed
Stemler (1982)	Anatomical correlates of emotion	False feedback	Study specific	Post induction	None detailed
Stiles & Götestam (1989)	Mood and positive thoughts	Velten	DACL	Pre and post induction	None detailed
Strack, Schwarz & Gschneidinger (1985)	Mood and memory	Autobiographical recall	Study specific	Post induction	None detailed
Sutton, Teasdale & Broadbent (1988)	Theories of depression	Music	VAS	Post induction	None detailed
Taylor & Cooper (1992)	Mood and self perception	Velten (adapted)	VAS	Pre and post induction	None detailed
Teasdale & Spencer (1984)	Mood and perceived success	Music	Study specific	Post induction	None detailed
Teasdale & Russell (1983)	Mood and memory	Velten	Study specific	Pre and post induction	None detailed
Teasdale & Fogarty (1979)	Mood and memory	Velten	Study specific	Pre and post induction	None detailed
Thompson, Cowan & Rosenhan (1980)	Mood and altruism	Story	Study specific	Post induction	15-60 minute therapy session
Tomarken, Davidson & Henriques (1990)	Neuroanatomy of emotion	Film	Study specific	Post induction	None detailed

Citation	Investigation	Induction technique	Mood assessment	Manipulation check	Mood removal
Van Rooijen & Vlaander (1984)	Induction technique	Film	DACL (translated)	Pre and post induction	Debrief and discussion
Varner & Ellis (1998)	Mood and cognition	Velten (adapted)	DACL	Post induction	E-VMIP statements
Velten (1968)	Induction technique	Velten	MAACL	Post induction	None detailed
Vrana & Lang (1990)	Mood and the startle response	Story	Study specific	Post induction	None detailed
Wenzlaff, Wegner & Klein (1991)	Mood and cognition	Music	Study specific	Post induction	Debrief
Wetzler (1985)	Mood and memory	Velten	DACL	Post induction	None detailed
Williams (1980)	Induction technique	Story	VAS	Post induction	Debrief - no details
Wiseman & Levin (1995)	Induction technique	Natural (relief from boredom)	PANAS	Post induction	None detailed
Wood, Saltzberg & Goldsamt (1990)	Mood and self perception	Autobiographical recall	VAS	Post induction	Debrief - no details
Wood, Saltzberg & Goldsamt (1990)	Mood and self perception	Music	VAS	Post induction	Debrief - no details
Wright & Mischel (1982)	Mood and social learning	False feedback	Nowlis MACL	Post induction	None detailed

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## Appendix 2

### The Velten Mood Induction Procedure Study

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#### A2.1 Instruction booklet

##### **Page 1:**

Please read each of the following pages to yourself. Then read each page out loud. Start with this page, but to avoid repetitiveness, begin with the statements below the line.

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I will read each of the following pages to myself. Then I will read each of the pages out loud. I won't worry about any reading errors which may occur.

##### **Page 2:**

I will be shown a series of statements on the computer in front of me. As each statement is presented to me, I will read it to myself, and then read it out loud. I'll go over each statement in my head as if it was one of my own thoughts. I will concentrate my full attention on the statements and exclude any unrelated thoughts.

##### **Page 3:**

If I feel the urge to laugh, it will probably be because humour is a good way to counteract unusual situations. I will try to avoid this kind of reaction.

If for any reason I feel I cannot continue, I will so indicate

## **A2.2 Elation VMIP statements**

I do feel pretty good today, though

I feel light hearted

This might turn out to be one of my good days

If your attitude is good, then things are good - and my attitude is good

I've certainly got energy and self confidence to spare

I feel cheerful and lively

On the whole, I have very little difficulty in thinking

My family are pretty proud of me most of the time

I'm glad I'm in university - it's the key to success nowadays

For the rest of the day, I bet things go really well

I'm pleased that most people are friendly to me

My judgement about most things is sound

It's encouraging that as I get further into my work, it's going to take less effort to do well

I'm full of energy and ambition - I feel like I could go a long time without sleep

This is one of those days where I can work with practically no effort at all

My judgement is keen and precise today - I'm feeling good

When I want to, I can make friends extremely easily

If I set my mind to it, I can make things turn out fine

I feel enthusiastic and confident now

There should be opportunity for a lot of good times coming along

My favourite song keeps going through my head

Some of my friends are so lively and optimistic

I feel talkative - I feel like talking to almost anybody



I'm full of energy, and I really like the things I'm doing in my life  
I'm able to do things accurately and efficiently  
I know good and well I can achieve the goals I set  
Most of the things that have depressed me in the past, wouldn't have if I'd just had the right attitude  
I have a sense of power and vigour  
I feel so vivacious and efficient today - sitting on top of the world  
It would really take something to stop me now  
In the long run, it's obvious that things have got better and better during my life  
I know in the future that I will not over emphasise so-called problems  
I'm optimistic that I can get along very well with most people I meet  
I'm too absorbed in things to have time to worry  
I'm feeling amazingly good today  
I'm particularly inventive and resourceful in this mood  
I feel superb! I think I can work to the best of my ability  
Things look good - things look great  
I feel that many of my friendships will stick with me in the future  
I can find good in almost anything  
I feel so playful today - I feel like surprising someone by telling a silly joke  
I feel exhilarated in all I do  
I feel highly perceptive and refreshed  
My memory is on good form today  
In a buoyant mood like this one, I can work fast and do it right the first time  
I can concentrate hard on anything I do  
My thinking is clear and rapid

Life is so much fun; it seems to offer so many sources of fulfilment

Things will get better and better today I can make decisions rapidly and correctly - and I can easily defend them against criticism

I feel industrious - I want to do something

Life is firmly in my control

I wish somebody would play some good loud music

This is great - I really do feel good and I am elated about things

I'm really feeling sharp now

This is just one of those days when I'm ready to go

I feel like bursting with laughter - I wish somebody would tell a joke and give me an excuse

I'm full of energy

I feel really great

### **A2.3 Depressed VMIP statements**

Today is neither better nor worse than any other day

However, I feel a little low today

I feel rather sluggish now

Sometimes I wonder whether university is all that worthwhile

Every now and then I feel so tired and gloomy that I'd rather just sit than do anything

I can remember times when everybody but me seemed full of energy

I have often found myself staring into the distance, my mind blank when I should be working

It has occurred to me more than once that study is useless because you forget everything you learn anyway

People annoy me, I wish I could be by myself  
I've had important decisions to make in the past, and I've sometimes made the wrong one  
I do feel somewhat discouraged and drowsy - maybe I'll have a nap when I get home  
Perhaps university takes more time, effort and money than it's worth  
I'm afraid the famine in Africa may get a lot worse  
I just don't seem to be able to get going as fast as I used to  
There have been days where I have felt weak and confused and everything went wrong  
Just a little bit of effort tires me out  
I've had daydreams where my mistakes keep happening to me - sometimes I wish I could start again  
I'm ashamed that I've caused my parents needless worry  
I feel terribly tired and indifferent to things today  
Just to stand up would take a big effort  
I'm getting tired out - I can feel my body getting exhausted and heavy  
I'm beginning to feel sleepy - my thoughts are drifting  
At times I've been so tired and discouraged that I went to sleep rather than face important problems  
My life is so tiresome - the same old thing day after day depresses me  
I couldn't remember things well right now if I had to  
I just cannot make up my mind - it's so hard to make simple decisions  
I want to go to sleep - I feel like just closing my eyes and going to sleep right here  
I'm not very alert - I feel listless and vaguely sad  
I've doubted I'm a worthwhile person  
I feel worn out - my health may not be as good as it's supposed to be  
It often seems that no matter how hard I try, things still go wrong

I've noticed that no one seems to really understand or care when I complain or feel unhappy

I'm uncertain about my future

I'm discouraged and unhappy about myself

I've laid awake at night worrying for so long that I've hated myself

Things are worse now than when I was younger

The way I feel now, the future looks boring and hopeless

My parents never really tried to understand me

Some very important decisions are almost impossible for me to make

I feel tired and depressed - I don't feel like working on things I know must get done

I feel horribly guilty about how I've treated my parents at times

I have a feeling that I just can't reach people

Things are easier and better for other people - I feel like there's no use in trying

Often people make me very upset - I don't like to be around them

It takes too much effort to convince people of anything - there's no point in trying

I fail to communicate with people about my problems

It's so discouraging the way people don't really listen to me

I've felt so alone before that I could have cried

Sometimes I've wished I could die

My thoughts are slow and down cast - I don't know what to think or say

I just don't care about anything - life just isn't fun

Life seems too much for me anyhow - my efforts are wasted

I'm so tired

I can't concentrate or move - I just want to forget about everything

I have too many bad things in my life

Everything seems utterly futile and empty  
I feel dizzy and faint - I need to put my head down and not move  
I don't want to do anything  
All the unhappiness of my past life is taking possession of me  
I want to go to sleep and never wake up

#### **A2.4 Neutral VMIP statements**

Seoul has the largest population in the world, with 10,229,262 people  
In 1995 there was a year long celebration marking the 50th anniversary of the United Nations  
At the end appears a section entitled "biography notes"  
There are two kinds of nouns denoting physical things: individual and mass  
The book or any part thereof must not be reproduced in any form  
Agricultural products comprised seventy percent of the income  
Saturn is sometimes in conjunction, behind the sun from the earth, and is not visible  
Some streets are said to be still listed under their old names  
The system is supervised by its own board of directors  
In Britain, the majority of roses are grown in the Channel Islands  
The EU milk subsidy helps provide children with free milk in British schools  
Natural selection is dependent on the heritable variations in traits  
The typography, paper and bind were of the highest quality  
The factory dominated the skyline for as long as anyone could remember  
The desk was old, and scratched into its surface was a profusion of dates and initials  
The Orient Express travels between London and Venice

When the Banyan tree bent down under its own weight, its branches began to take root  
There isn't a scientific explanation for every UFO sighting  
The Hope diamond was shipped from South Africa to London through the normal mail service  
The review was concerned with the first three volumes  
The ship was ancient, and would soon retire from the fleet  
Slang is a constantly changing part of the language  
An article in the local newspaper indicates the acceptance of the kidnapper's terms  
There are some situations where no oath is required  
The Guardian's Soulmates tries to bring lonely people together  
99.1% of Alaska is owned by the Federal Government  
Two men dressed as repairmen will appear shortly after the van pulls up  
The wood was discoloured, as if it had been held in a fire  
Someone noticed a light in the darkness, it was just above the horizon  
Painting in non-European countries is covered in a separate volume  
A survey revealed that there has been a 40% decline in traditional graduate jobs  
Provoked arousal is accompanied by steep negative shifts  
The names on the Christmas mailing list are alphabetically ordered  
Significantly, these changes occurred in the full moon  
West Samoa gained independence in 1965  
The magazine report was biased, as normal  
The map would prove useless as a beginners guide  
The speaker outlined a plan where deficits would be eliminated  
The black and white pictures are arranged in ten sections  
No part of the United Kingdom is more than 75 miles from the sea

It has been on the front pages for days

The notice made it clear that coffee breaks were limited

No man worked harder than he

Canary Wharf tower is the tallest building in Britain

Tesco has been voted Britain's most admired company

The doorkeeper was dressed in red

During the next ten years, the group participated in politics

The organisation depended on volunteers for support

More than a billion drinks made by the Coca-Cola company are consumed each day

It was their sixth consecutive best seller

It all fitted in with the officer's story

The merger did not alter the company's policy

The mansion was rented by the committee

There are over 3500 trade associations and professional bodies in the UK

Utah is known as the beehive state

After the accident, changes were made to the transportation arrangements

The Chinese language has many dialects - including Mandarin, Cantonese and Wu

Fordwich, in Kent, is the smallest town in Britain

At low tide, the hull of the ship could be seen

A free sample will be given to each person who enters the store

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## Appendix 3

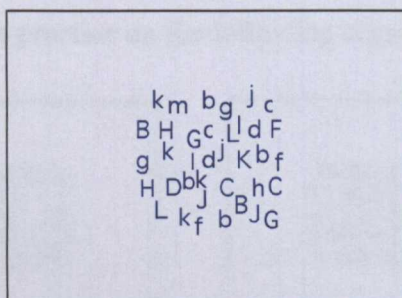
### The False Feedback Mood Induction Procedure Study

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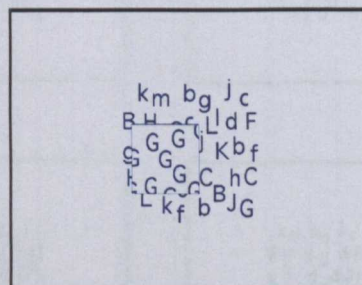
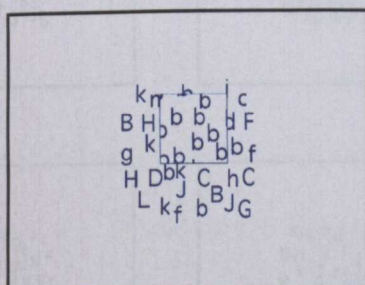
#### A3.1 Instructions

You will be performing a verbal working memory task. The main aim is to memorise a series of letters and compare them to letters that have appeared previously in the series. In this particular case you will be comparing each letter to the letter which appeared three trials previously.

The letters will be presented to you on the computer screen on the following background:

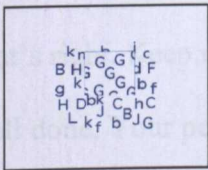


They can occur in any position over the above background and may be in upper or lower case. For example:

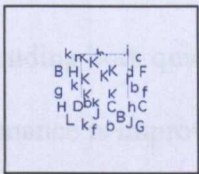




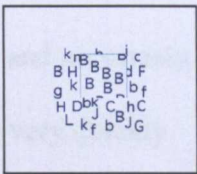
Should a letter be the same as that three trials previously, you will press ‘s’:



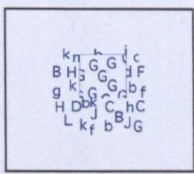
Trial 1



Trial 2

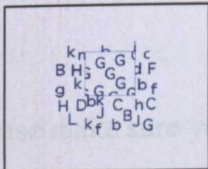


Trial 3

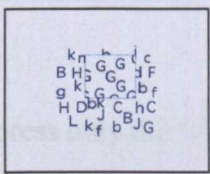


Trial 4

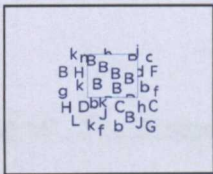
Should a letter be different to that three trials previously, you will press ‘d’:



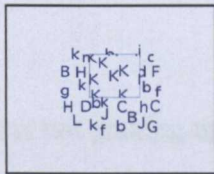
Trial 1



Trial 2

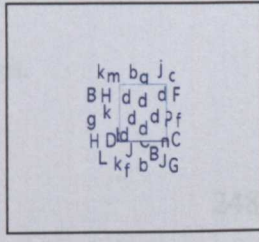
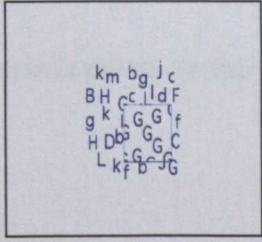
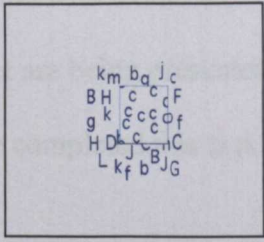
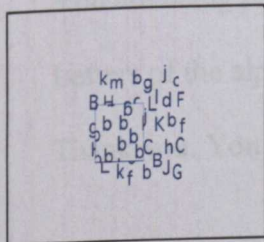
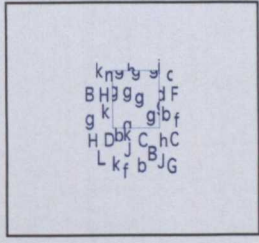
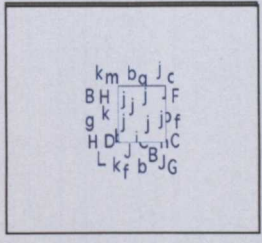
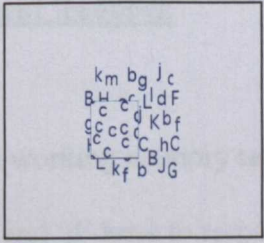
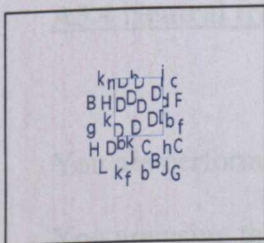
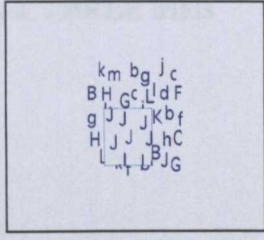
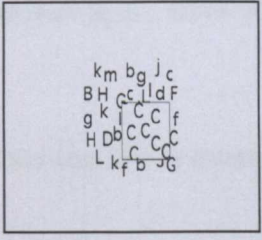
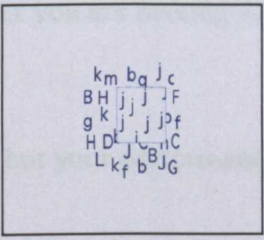
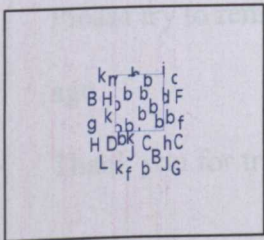


Trial 3



Trial 4

Before you begin, let’s have a practice on the following sequence of letters:



### **A3.2 Elation treatment feedback**

That's right. Keep responding both quickly and accurately.

Well done, Your performance is improving very quickly.

You are now performing at a level above average.

You must be thinking very clearly. Your performance is excellent.

Thank you. You performed very well on this task.

### **A3.3 Depression treatment feedback**

Please make sure you press only the 's' and 'd'. The computer is not picking up all of your responses.

Please try to make sure your answers are both accurate and fast.

Your performance has fallen below average. Please concentrate on the task.

Please try to remember you are making a comparison to the letter you saw THREE trials ago.

Thank you for trying but your performance did not rise above average.

### **A3.4 Neutral treatment feedback**

You are performing a working memory task.

You are using the 's' and 'd' keys to respond.

You are being presented with letters of the alphabet

Letters of the alphabet are being presented at various points on the screen.

Thank you. You have completed this task.

---

## **Appendix 4**

### **The Biographical Recall Mood Induction Procedure Study**

---

#### **A4.1 Elation treatment**

##### **Slide 1:**

I would now like to ask you to take a few minutes to look into your past and think what have been the two happiest events of your life.

When you have finished reading these instructions, take ten minutes to think of these events.

I would like you to try and think of all the details of what was happening at the time, to the point that you could imagine them happening to you now.

##### **Slide 2:**

Think about how old you were, who were the people or events involved, and what your feelings were.

You will hear a beep when the 10 minutes are over and you will be asked a few questions related to the images you thought of.

It is very important that you take this reflection task very seriously. Think of those events that made you feel as if you were on top of the world and had everything going for you.

**Slide 3:**

Please sit back, close your eyes, and relax.

Take your time and think about the happy events.

**Slide 4:**

Please start when you hear the beep.

You will hear a second beep when the 10 minutes are over and you will then be shown the questions.

**Questions**

1. Can you remember how old you were when these events happened?
2. Can you remember where these events happened?
3. Can you remember anybody else who was involved in these events?

**A4.2 Depression treatment**

**Slide 1:**

I would now like to ask you to take a few minutes to look into your past and think what have been the two saddest events of your life.

When you have finished reading these instructions, take ten minutes to think of these events.

I would like you to try and think of all the details of what was happening at the time, to the point that you could imagine them happening to you now.

**Slide 2:**

Think about how old you were, who were the people or events involved, and what your feelings were.

You will hear a beep when the 10 minutes are over and you will be asked a few questions related to the images you thought of.

It is very important that you take this reflection task very seriously. Think of those events that made you feel lonely, rejected, defeated or hurt.

**Slide 3:**

Please sit back, close your eyes, and relax.

Take your time and think about the unhappy events.

**Slide 4:**

Please start when you hear the beep.

You will hear a second beep when the 10 minutes are over and you will then be shown the questions.

**Questions**

1. Can you remember how old you were when these events happened?
2. Can you remember where these events happened?
3. Can you remember anybody else who was involved in these events?

---

# Technical Appendix 1

## BTEC Clinical Neurophysiology

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### TA1.1 Course Details

The course was undertaken at the City of Westminster College, London over the 2000/2001 academic year. The Clinical Neurophysiology course is a module of the HND in Medical Physics and Physiological Measurement, which can be taken as a stand alone BTEC Professional Development Module.

### AT1.2 Course Syllabus

- **Anatomy and physiology; pathology and treatment**
  - With respect to neurological embryology, brain, brainstem, spinal cord, peripheral nerves, muscle
- **Measurements, techniques, applications, instrumentation principles, recording practice**
  - electroencephalography – awake, sleep, monitoring, telemetry,
  - activation procedures (sleep/hyperventilation/photic-stimulation),
  - electromyography,
  - nerve conduction,
  - evoked potentials – visual, auditory, somatosensory, motor, event-related, retinal, cochlear,
  - eye movements, other effects (age/awareness/drugs)

- **Neuro-imaging techniques and applications**
  - MRI, PET, CT, Doppler, DSA
- **Applied psychiatry**
  - disability, psychoses, neuroses, personality, behaviour

### **TA1.3 Course Aims**

The aim of the module is to provide the student with a sufficient level of specialised clinical neurophysiological knowledge to allow a high degree professionalism, facilitate promotion to the higher MTO grades, and enable contribution to the specialised education of trainee staff, enable acquisition of ECNE Board Part II exam.

### **TA1.4 Course Objectives**

- To be able to demonstrate discipline-specific workplace skills to a high degree of competence in the clinical neurophysiological clinical environment.
- To be able to contribute to the ensurance of quality in the measurement function of the clinical physiological measurement department.
- To achieved the basic level of Professional Body accreditation / examination (ECNE Board Part I).
- To be working towards the higher level Professional Body accreditation / examination (ECNE Board Part II)

### **TA1.5 Achievement**

**BTEC Clinical Neurophysiology (Merit)**

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## **Technical Appendix 2**

### **Electrooculogram correction of electroencephalographic data**

---

#### **TA2.1 General Introduction**

During the interpretation of the EEG and its derivatives, there are a number of negative factors that must be considered. Foremost is the point of measurement will not indicate the location of the neuronal generator, due to the conductive properties of neuronal tissue, the skull and scalp. However, equally detrimental is the degree to which electrical activity generated by eye movements contributes to the measured neural potentials.

The ways in which the contamination of neural potentials by ocular potentials can lead to the misinterpretation of data are manifold (Croft & Barry, 2000a). For example, ocular potentials can make a significant contribution to event-related phenomenon such as contingent negative variation (CNV; (Low, Borda, Frost, & Kellaway, 1996; Hillyard & Gallambos, 1970). Also, spectral power analysis of the EEG can be corrupted by ocular potentials increasing the power of low frequency oscillations (Gasser, Ziegler, & Gattaz, 1992).

Picton's et al (2000) guidelines for publishing event-related potential studies highlights the importance which should be placed upon the observation of ocular potentials:



“It is essential to monitor ocular artefacts using electrodes near the eyes when recording most ERPs.” ((Picton et al., 2000a); p137)

Further, the guidelines show that the discussion over whether it is better to correct for ocular artefacts or reject data contaminated with ocular artefacts is rife:

“Although rejection procedures can be used to eliminate artefacts in many normal subjects, these protocols will not be satisfactory if the artefacts are very frequent” ((Picton et al., 2000a); p138)

### **TA2.2 The Origin of Ocular Contamination**

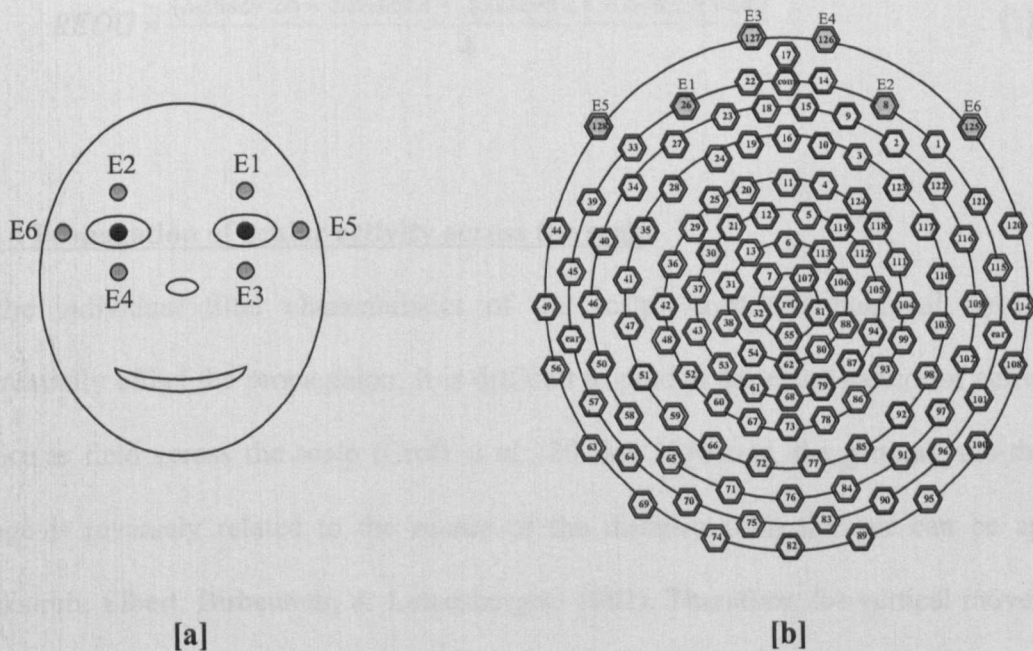
There are a number of generators which contribute to the electrical field which surrounds the eye, the dominant one being the cornea-retinal dipole (Croft et al., 2000a). The cornea is positive and the retina is negative, resulting in the eye itself forming an electrical dipole. Therefore any movement of the eye, whether vertical, horizontal or oblique, will cause electrical field fluctuations which will interact with the fields surrounding neuronal source generators (Croft et al., 2000a). As a second dipole exists across the retina alone, eye movements also contribute a posterior source generator (Croft et al., 2000a).

The final contributor is the action of the eyelid sliding across the eye (Croft et al., 2000a), where the sliding motion alone is capable generating a potential change (Matsuo, Peters, & Reilly, 1975a). Therefore there are three spatially distinct generators which interact together to produce the complex ocular field.

**TA2.3 Determination of Ocular Activity**

The most widely used measurement tool is to place a series of electrodes round the eyes in order to record the electrooculogram (EOG;(Croft et al., 2000a)). The EOG montage places electrodes above, below and at the outer canthi of each eye all recording to a common reference, however the distribution of the sensors on the geodesic sensor net allows comparable recording to be made (Figure TA2.1).

To account for the differential effect of the source generators associated with the ocular field, three orthogonal values can be derived from the EOG: vertical EOG (VEOG), horizontal EOG (HEOG) and radial EOG (REOG) (Elbert, Lutzenberger, Rockstroh, & Birbaumer, 1985).



**Figure TA2.1** Measuring the electrooculogram using [a] traditional electrode placement and [b] a geodesic sensor net.

To calculate the VEOG, the following equation is used (Croft & Barry, 2000c):

$$VEOG = \frac{((Sensor26 + Sensor8) - (Sensor127 + Sensor126))}{2} \quad (1)$$

As it is assumed that the same amount of neuronal potential will reach each of the EOG sensors, the subtraction of sensor 8 from 26 and sensor 126 from 127 ensures the correct proportion of neuronal activity is reflected in the VEOG (Croft et al., 2000a). The calculation of the HEOG uses the following equation (Croft et al., 2000c):

$$HEOG = Sensor128 - Sensor125 \quad (2)$$

While the REOG is calculated using (Croft et al., 2000c):

$$REOG = \frac{(Sensor26 + Sensor8 + Sensor127 + Sensor128)}{4} \quad (3)$$

#### **TA2.4 Propagation of ocular activity across the scalp**

As the individual filter characteristics of the scalp, skull and neuronal tissue will differentially effect the propagation, it is difficult to predict the exact pattern of activity of the ocular field across the scalp (Croft et al., 2000a). However, the general rule that the voltage is inversely related to the square of the distance from the eye can be applied (Rockstroh, Elbert, Birbaumer, & Lutzenberger, 1982). Therefore, for vertical movements it would be expected that 0.2 of ocular activity would be present at frontal sites, 0.05 present at occipital sites, and that there would be little influence on lateral sensors (Croft et al., 2000a). Alternatively, horizontal movements would produce similar effects on frontal

and occipital sites but the lateral propagation would be greater. Sensors on the left side of the head would be positively effected while those on the right would be negatively effected, leaving midline sensors with little or no ocular activity (Croft et al., 2000a).

## **TA2.5 Dealing with data contaminated by ocular artefacts**

### **TA2.5.1 Alterations of methodological procedures**

It appears that the obvious way to avoid the ocular contamination of EEG recordings is to simply acquire data with the participant's eyes closed (Croft et al., 2000a). Unfortunately, this method causes more problems than if the participant's eyes were open. Not only would there be strict limitations placed on the kinds of investigations that could be conducted, but there would also be more subtle electrophysiological consequences. Ocular contamination may still occur as, without the presence of a visual reference point, there may be an increase in low frequency eye movements (Croft et al., 2000a). Also, there would be non-task specific alterations to the EEG power spectrum as eye closure would inhibit alpha blocking (Croft et al., 2000a).

The next option would be to control the participant's creation of eye movements and blinks (Croft et al., 2000a). This could be done in a number of ways: encouraging the participant to focus on a fixation point (Hillyard et al., 1970), instructing the participant to not to blink when the EEG is being recorded, or instructing them to blink only between trials or when a 'blink now' cue is given. Again, this approach is accompanied by a number of difficulties.

By instructing the participant to focus on the production of eye blinks and movements, a dual-task scenario is automatically included into the experimental paradigm (Croft et al.,

2000a). Not only may this have a detrimental effect on cognitive processes and behavioural data, it has been shown to influence ERP morphology (Verleger, 1991; Weerts & Lang, 1973). Further, this approach may place restrictions upon the participant populations that may be included in studies as there may be difficulties associated with task compliance, for example schizophrenic patients (Croft et al., 2000a). Finally, the simple act of focusing the participant's attention upon their blinking and eye movement production may result in an increase in both activities.

#### **TA2.5.2 Rejecting data contaminated by ocular artefacts**

If the production of eye blinks and movements cannot be avoided, then the logical step is to omit trials containing ocular contamination during the signal processing stage of EEG/ERP data manipulation (Croft et al., 2000a). This is the method used by the NetStation software. However, even when strict rejection criteria are applied to data, ocular artefacts could still be present in the data (Croft et al., 2000a). Trials containing small eye movements or the beginning/end of blinks could escape the detection process and increase the risk of data misinterpretation (Rowland, 1968). Also, by rejecting trials contaminated by blinks and eye movements, valuable experimental data may be lost (Croft et al., 2000a).

Secondly, it is possible that certain clinical populations may exhibit different types or levels of eye movements and blinks (Croft et al., 2000a). Where this is the case, the removal of contaminated trials would lead to a design bias. Thirdly, should the neurological signature of a cognitive or perceptual process be coupled to ocular activity then it would be overlooked (Croft et al., 2000a). For example a physiological reaction such as the startle response would be impossible to investigate as a result of its temporal connection with a blink (Putnam & Roth, 1990). Finally, the rejection of data severely decreases the

amount of data which can be included in the statistical analysis of EEG/ERP data therefore reducing statistical power.

### **TA2.5.3 Correcting data contaminated by ocular artefacts**

The final way of dealing with the ocular contamination of data is to filter out the ocular potentials from the neural potentials recorded at the scalp. To do this, the proportion of ocular activity present at each recording site must be precisely calculated in order for the artefact to be removed from the EEG without modifying the relevant neuronal activity.

#### **TA 2.5.3.1 Analogue techniques**

The main problem with the earliest correction techniques was the level of sensitivity obtained during the estimation of ocular artefacts (Croft et al., 2000a). For example, one such technique relied upon the visual identification of ocular contaminated data (Girton & Kamiya J, 1973a). Unsurprisingly, such analogue techniques are inferior to the mathematical algorithms employed today.

#### **TA2.5.3.2 Mathematical techniques**

The simplest correction of EEG data would involve the subtraction of the EOG signal from each trial, so that the portion of EEG attributed to ocular activity is removed (Gratton, Coles, & Donchin, 1983b). However the EOG signal will be also be susceptible to volume conduction across the skull and therefore the amount of contamination will vary across the sensors. Therefore the subtraction of the same EOG signal from each sensor would equally result in a contaminated EEG signal (Gratton et al., 1983b). Therefore the general aim of mathematically derived correction algorithms can be expressed as:

$$\text{True EEG} = \text{Measured EEG} - \text{B.EOG}$$

where  $B$  is the proportion of ocular activity contaminating the measure EEG signal. Therefore the comparison of correction procedures is centred upon the accuracy of the  $B$  coefficient.

Contemporary eye movement correction techniques derive correction factors from the mathematically determined source of the EOG signal, rather than the recorded ocular potentials (Matsuo, Peters, & Reilly, 1975b; Lins, Picton, Berg, & Scherg, 1993; Berg & Scherg, 1991; Berg & Scherg, 1994; Ille, Berg, & Scherg, 1997). Using principal component analysis (Ille et al., 1997) or dipole models (Berg et al., 1994; Lins et al., 1993), coefficients are calculated by combining the source characteristics of each type of eye movement. On first consideration, this appears to be a technically superior method of coefficient computation. Advantages include factors such as the source of ocular activity will be free of contaminants, the activity associated with each type of eye movement can be clearly differentiated, and the sensors normally used in the measurement of the EOG can contribute to the EEG recording (Picton et al., 2000b).

Unfortunately the procedure is associated with a major drawback as the presumed precision is dependent upon the accuracy of the model used to determine the source of the ocular potentials (Croft & Barry, 2000b). The successful application of source localisation algorithms requires the stereotaxic co-ordinates of the sensor position to be co-registered with the individual's MRI scan. Although the digitisation of sensor position is relatively easy, the cost of and access to MRI scanning is still prohibitive to many. However as the solutions to the inverse problem become more sophisticated, the digitisation of sensor positions alone will be sufficient for the execution of source localisation algorithms.

Consequently, traditional eye movement correction techniques rely upon regression procedures to calculate the B coefficient (Croft et al., 2000b). The two main techniques employed in electrophysiological research are the Eye Movement Correction Procedure (EMCP; (Gratton et al., 1983b) and Aligned-Artifact Average algorithm (AAA; (Croft & Barry, 1998). The comparison of these two techniques is based upon their ability to deal with the main concern surrounding the calculation of B coefficients, namely can the same coefficient can be used for all types of eye movement.

For the same B coefficient to be used in the correction of horizontal, vertical and radial eye movements, the propagation of these signals across the scalp must be identical. Although a number of studies have identified that the ocular potentials generated by blinks and saccades have distinct characteristics (Girton & Kamiya J, 1973b; Corby & Kopell, 1972; Lins et al., 1993; Overton & Shagass, 1969), there is disagreement as to whether this strict criteria is necessary.

Gratton et al (1983) argue that the additional eyelid movement that is associated with blinks leads to the generation of an electrical field that is characteristically different from eye movements. Therefore to avoid movement artefacts remaining in the data, separate coefficients should be calculated (Gratton et al., 1983b). However, Croft & Barry (1998) feel this viewpoint reflects the inadequacy of older techniques rather than it being a consequence of electrophysiological characteristics, and concludes a more accurate correction is achieved when a single coefficient is employed.



## **TA2.6 Conclusion**

As this argument is not competently resolved, the main distinguishing feature between these two techniques is the situation under which the EOG signal used to create the coefficient is recorded. A major concern surrounding the AAA technique is the validity of recording calibration eye movements prior to the experimental session. Croft & Barry (1998) argue that risk of the fluctuations in the propagation of ocular potentials during the recording session or there being a significant difference between the characteristics of eye movements recorded during both session is minimal. However, the existence of differences in the electrophysiological characteristics of voluntary and involuntary eye movements (Gratton, Coles, & Donchin, 1983a) points to erring on the side of caution. Therefore the eye movement correction procedure design by Gratton, Coles & Donchin (1983) will be implemented in this thesis.

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## **Technical Appendix 3**

### **A Signal Processing Toolbox for MatLab**

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#### **TA3.1 Development of a Signal Processing Toolbox**

The first release of NetStation was associated with a number of problems, including the inability to concatenate recordings made on separate sessions. Also, the graphing capabilities were limited as the emphasis had been placed upon the acquisition of data during the development of version 1. A further problem was that the system's inbuilt method of dealing with EOG contamination was the rejection of data.

At this point in time there was a limited amount of third party software that could deal with the NetStation file format, therefore it was decided to develop a signal processing toolbox for MatLab (Mathworks, Inc). Once EEG data had been digitally filtered, segmented and assessed for quality within NetStation, it was exported in the MatLab format and all subsequent signal processing was performed by this toolbox.

There are four components to the toolbox: the preparation of data and reference files, eye movement correction, signal processing and plotting functions.

## **TA3.2 Data preparation and reference files**

### **TA3.2.1 RENAME\_\***

**Variations:** rename\_P300 (used as example); rename\_stroop; rename\_wm

```
% RENAME_P300 Renames default variable names
%
% Segments exported from NetStation are given the default name of
% CategoryXSegmentN. RENAME changes the variable name to StandardN for
% Category1 variables and TargetN for Category2 variables.

% where N is the number of Standard variables
Standard1 = Category1Segment1; clear Category1Segment1
...
StandardN = Category1SegmentN ; clear Category1SegmentN

% where N is the number of Target variables
Target1 = Category2Segment1; clear Category2Segment1
...
TargetN = Category2SegmentN; clear Category2SegmentN
```

### **TA3.2.2 CONCATENATE\_\***

**Variations:**

Participant files - concatenate\_p300 (used as example); concatenate\_stroop;  
concatenate\_wm;  
Experiment files - concatenate\_subjects

```
% CONCATENATE_P300 Creates 3D array of P300 data
%
% Concatenation of individual trials from a P300 study to form three
% dimensional arrays for standard and target stimuli

% STANDARD TRIALS - ammend filename: <S#Cstandards> (where: S# = subject
% number, C = condition), where N is the number of Standard variables

S#Cstandards = cat(3,Standard1, ... StandardN);
clear Standard*

% TARGET TRIALS - ammend filename: <S#Ctargets> (where: S# = subject
% number, C = condition), % where N is the number of Target variables

S#Ctargets = cat(3,Target1, ... TargetN);
clear Target*
```

### **TA3.2.3 BAD\_DATA**

```

% BAD_DATA Script to assign bad channels and segments
%
% Using the information gained from the NetStation 'moving window'
% function, bad_data creates the cell array BAD{[seg],[channels]}. [seg]
% is a single number which indicates the segment classification (1|0,
% where 1 = good; 0 = bad segment). [channels] is a double array which
% indicates the channel classification for that segment (1 = good; 0 =
% bad). BAD is a reference array during signal processing functions.

% >> add number of segments:
no_dim = 144;
% >> add number of channels:
no_rows = 129;
% >> add number of samples:
no_columns = 250;

bad = cell(no_dim,2);
for i = 1:no_dim
    bad{i,1} = ones;
    bad{i,2}(1:no_rows,1:no_columns) = ones;
end

% >> add list of bad segments:
bad_segments = [];
for j = 1:length(bad_segments)
    bad{(bad_segments(j)),1} = 0;
end

% >> add list of channels which are bad across all segments:
bad_channels = [];
for i = 1:no_dim
    for j = 1:length(bad_channels)
        if bad{i,1} == 1
            bad{i,2}((bad_channels(j)), :) = 0;
        else
            bad{i,2} = [];
        end
    end
end

% add bad channels in individual segments ([segment],[channelno];):
bad_channel_array = {[],[],...};
for i = 1:length(bad_channel_array)
    for j = 1:length(bad_channel_array{i,2})
        bad{(bad_channel_array{i,1}),2}((bad_channel_array{i,2}(j)), :) = 0;
    end
end

% TO BE RUN IF NO BAD CHANNELS ALL SEGMENTS
%for i = 1:no_dim
%    if bad{i,1} == 0
%        bad{i,2} = [];
%    end
%end

```

### TA3.2.4 AV\_BAD\_DATA

```

% AV_BAD_DATA Bad data reference file (averaged over subjects)
%

```

```

% Creates cell array AV_BAD{[plane], [channels]}. [plane] - refers to
% subject and [channels] - is a double array(channel,sample) which refers
% to channels which are bad for the averaged data where:1 = good sample;
% 0 = bad sample. AV_BAD used as reference array when any function is
% applied to data averaged across subjects

% >> add number of subjects:
no_dim = 8;
% >> add number of channels:
no_rows = 129;
% >> add number of samples:
no_columns = 250;

global av_bad
av_bad = cell(no_dim,2);
    for i = 1:no_dim
        av_bad{i,1} = i;
        av_bad{i,2}(1:no_rows,1:no_columns) = ones;
    end

% >> add bad channel information for subjects ({[plane],[channel];...}):
bad_data_array = {[],[]; ...};

for i = 1:length(bad_data_array)
    for j = 1:length(bad_data_array{i,2})
        av_bad{(bad_data_array{i,1}),2}((bad_data_array{i,2}(j)), :) = 0;
    end
end

clear no_dim; clear no_rows; clear no_columns; clear bad_data_array;
clear i; clear j;

```

### **TA3.3 Eye Movement Correction**

#### **TA3.3.1 \*EOG\_CORRECT**

##### **Variations:**

**VEOG\_CORRECT:** Example given below

**HEOG\_CORRECT:** As above but using the following heog calculation –

```

% Calculation of horizontal eye movements from sensors 128 and 125 of the
% GSN, adapted from using Croft & Barry (2000)
heog(1:no_trials,1:no_samples) = zeros;
for i = 1:no_trials
    if bad{i,1} == 1
        heog(i,:) = x(128,:,i)-x(125,:,i);
    end
end

```

end

**REOG\_CORRECT:** As above but using the following reog calculation -

```

% Calculation of radial eye movements from sensors 26, 8, 126 and 127 of
% the GSN, adapted from using Croft & Barry (2000)
reog(1:no_trials,1:no_samples) = zeros;

```

```

for i = 1:no_trials

```

```

        if bad{i,1} == 1
            reog(i,:) = (x(26,:,i)+x(8,:,i)+x(127,:,i)+x(126,:,i))./4;
        end
    end
end

```

**NB** The appropriate BAD file should be loaded into the workspace and declared a global variable prior to running function.

```

function [y,b] = VEOG_correct (x, t)
% VEOG_CORRECT corrects ERP data for vertical ocular voltage
%
% Takes 3D array X (representing the trial data for 1 participant over
% one condition) and corrects for vertical ocular voltage (VEOG) in the
% trials listed in T, using the EMCP created by Gratton, Coles & Donchin
% (1983).

global bad

no_channels = size(x,1);
no_samples = size(x,2);
no_trials = size(x,3);

% Calculation of vertical eye movements from sensors 26, 8, 126 and 127 of
% the GSN, adapted from using Croft & Barry (2000)

veog(1:no_trials,1:no_samples) = zeros;
for i = 1:no_trials
    if bad{i,1} == 1
        veog(i,:) = ((x(26,:,i)+x(8,:,i))-(x(127,:,i)+x(126,:,i)))./2;
    end
end

total_veog(1,no_samples) = zeros;
for i = 1:no_trials
    if bad{i,1} == 1
        total_veog(1,:) = total_veog(1,:) + veog(i,:);
    end
end

no_veogs(1,1) = zeros;
for i = 1:no_trials
    no_veogs(1,1) = no_veogs(1,1) + bad{i,1};
end

av_veog(1,1:no_samples) = zeros;
for i = 1:no_samples
    av_veog(1,i) = total_veog(1,i)./no_veogs;
end

sub_veog(1:no_trials,1:no_samples) = zeros;
for i = 1:no_trials
    if bad{i,1} == 1
        sub_veog(i,:) = veog(i,:) - av_veog;
    end
end

total_eeg(no_channels,no_samples) = zeros;
for i = 1:no_channels
    for j = 1:no_samples

```

```

        for k = 1:no_trials
            if bad{k,1} == 1
                if bad{k,2}(i,j) == 1
                    total_eeg(i,j) = total_eeg(i,j) + x(i,j,k);
                end
            end
        end
    end
end

no_eegtrials(no_channels,no_samples) = zeros;
for i = 1:no_channels
    for j = 1:no_samples
        for k = 1:no_trials
            if bad{k,1} == 1
                no_eegtrials(i,j) = no_eegtrials(i,j) + bad{k,2}(i,j);
            end
        end
    end
end

av_eeg(no_channels,no_samples) = zeros;
for i = 1:no_channels
    for j = 1:no_samples
        if no_eegtrials(i,:) ~= 0
            av_eeg(i,j) = total_eeg(i,j)./no_eegtrials(i,j);
        end
    end
end

sub_eeg(1:no_trials,1:no_samples,1:no_channels) = zeros;
for i = 1:no_channels
    for j = 1:no_trials
        if bad{j,1} == 1
            if bad{j,2}(i,:) == 1
                sub_eeg(j,:,i) = x(i,:,j) - av_eeg(i,:);
            end
        end
    end
end

sub_numerator_b(1:no_trials,1:no_samples,1:no_channels) = zeros;
for i = 1:no_channels
    for j = 1:no_samples
        for k = 1:no_trials
            if bad{k,1} == 1
                if bad{k,2}(i,:) == 1
                    sub_numerator_b(k,j,i) = sub_veog(k,j).*sub_eeg(k,j,i);
                end
            end
        end
    end
end

numerator_b(1,no_channels) = zeros;
for i = 1:no_channels
    numerator_b(1,i) = sum(sum(sub_numerator_b(:,:,i)));
end

```

```

sub_denominator_b(1:no_trials,1:no_samples,1:no_channels) = zeros;
for i = 1:no_channels
    for j = 1:no_samples
        for k = 1:no_trials
            if bad{k,1} == 1
                if bad{k,2}(i,:) == 1
                    sub_denominator_b(k,j,i) = sub_veog(k,j).*sub_veog(k,j);
                end
            end
        end
    end
end

denominator_b(1,no_channels) = zeros;
for i = 1:no_channels
    denominator_b(1,i) = sum(sum(sub_denominator_b(:,:,i)));
end

b(1,no_channels) = zeros;
for i = 1:no_channels
    if numerator_b(1,i) ~= 0
        b(1,i) = numerator_b(1,i)./denominator_b(1,i);
    end
end

prop_veog(1:no_channels,1:no_samples,1:length(t)) = zeros;
for i = 1:length(t)
    for j = 1:no_channels
        for k = 1:no_samples
            if bad{t(i),1} == 1
                if bad{t(i),2}(j,:) == 1
                    prop_veog(j,k,i) = b(1,j).*veog(t(i),k);
                end
            end
        end
    end
end

prop_eeg(1:no_channels,1:no_samples) = zeros;
for i = 1:no_channels
    for j = 1:no_samples
        prop_eeg(i,j) = av_eeg(i,j).*b(1,i);
    end
end

C(1:no_channels,1:no_samples) = zeros;
for i = 1:no_channels
    C(i,:) = av_veog(:,i) - prop_eeg(i,:);
end

for i = 1:length(t)
    for j = 1:no_channels
        if bad{t(i),1} == 1
            if bad{t(i),2}(j,:) == 1
                x(j,:,t(i)) = x(j,:,t(i)) - prop_veog(j,:,i) - C(j,:);
            end
        end
    end
end
end

```



```
y(1:no_channels,1:no_samples,1:no_trials) = zeros;
y = x;
```

## **TA3.4 Signal processing**

### **TA3.4.1 REREFERENCE**

**NB** The appropriate BAD file should be loaded into the workspace and declared a global variable prior to running function.

```
function y = rereference(x)
% REREFERENCE rereferences ERP data to the average voltage
%
% Re-references participant file 'X' using the Method 2 of the EGI
% Technical Note, 8/21/00. In each segment, the average voltage
% during each time sample is calculated for each electrode (uses a
% 129th row of zeros, which represent the voltage of the reference
% electrode relative to itself) and is then subtracted from the
% recording to produced the re-referenced file. Requires appropriate
% BAD reference file

global bad
no_rows = size(x,1);
no_columns = size(x,2);
no_dim = size(x,3);

total= zeros(no_dim, no_columns);
for i = 1:no_dim
    for j = 1:no_rows
        for k = 1:no_columns
            if bad{i,1} == 1
                if bad{i,2}(j,k) == 1
                    total(i,k) = total(i,k) + x(j,k,i);
                end
            end
        end
    end
end

no_channels = zeros(no_dim,no_columns);
for i = 1:no_dim
    if bad{i,1} == 1
        no_channels(i,:) = sum(bad{i,2});
    end
end

av_voltage = zeros(no_dim,no_columns);
for i = 1:no_dim
    if bad{i,1} == 1
        av_voltage(i,:) = total(i,:)./no_channels(i,:);
    end
end

y = zeros(no_rows,no_columns,no_dim);
```

```

for i = 1:no_dim
    for j = 1:no_rows
        if bad{i,1} == 1
            if bad{i,2}(j,:) == 1
                y(j,:,i) = x(j,:,i) - av_voltage(i,:);
            end
        end
    end
end
end

```

### TA3.4.2 BASELINE\_CORRECT

**NB** The appropriate BAD file should be loaded into the workspace and declared a global variable prior to running function.

```

function y = baseline_correct(x, t)
% BASELINE_CORRECT baseline corrects ERP data
%
% Calculates the mean voltage for each channel during specified time
% period, and subtracts this from each channel. Run using the command
% BASELINE_CORRECT(X,T), where X is the data set and T is the sample
% period which refers the duration of the baseline recording.

global bad
no_rows = size(x,1);
no_columns = size(x,2);
no_dim = size(x,3);
total_baseline = zeros(no_rows,no_dim);
for i = 1:no_rows
    for j = 1:no_dim
        for k = 1:length(t)
            if bad{j,1} == 1
                if bad{j,2}(i,:) == 1
                    total_baseline(i,j) = total_baseline(i,j) + x(i,(t(k)),j);
                end
            end
        end
    end
end

average_baseline = zeros(no_rows,no_dim);
for i = 1:no_rows
    for j = 1:no_dim
        if bad{j,1} == 1
            if bad{j,2}(i,:) == 1
                average_baseline(i,j) = total_baseline(i,j)./length(t);
            end
        end
    end
end

y = zeros(no_rows,no_columns);
for i = 1:no_rows
    for j = 1:no_columns
        for k = 1:no_dim
            if bad{k,1} == 1
                if bad{k,2}(i,:) == 1

```

```

                                y(i,j,k) = x(i,j,k) - average_baseline(i,k);
                                end
                        else
                                y(i,j,k) = 0;
                        end
                end
        end
end

```

### TA3.4.3 AVERAGE

**NB** The appropriate BAD file should be loaded into the workspace and declared a global variable prior to running function.

```

function y = average(x)
% AVERAGE averages across single trial data
%
% Takes the 3D array X (representing the trial data for 1 participant
% over 1 condition) and produces the average voltage across all channels
% in a double array

global bad

no_rows = size(x,1);
no_columns = size(x,2);
no_dim = size(x,3);

total_voltage(no_rows,no_columns) = zeros;
for i = 1:no_rows
    for j = 1:no_columns
        for k = 1:no_dim
            if bad{k,1} == 1
                if bad{k,2}(i,j) == 1
                    total_voltage(i,j) = total_voltage(i,j) + x(i,j,k);
                end
            end
        end
    end
end

no_trials(no_rows,no_columns) = zeros;
for i = 1:no_rows
    for j = 1:no_columns
        for k = 1:no_dim
            if bad{k,1} == 1
                no_trials(i,j) = no_trials(i,j) + bad{k,2}(i,j);
            end
        end
    end
end

y(no_rows,no_columns) = zeros;
for i = 1:no_rows
    for j = 1:no_columns
        if no_trials(i,:) ~= 0
            y(i,j) = total_voltage(i,j)./no_trials(i,j);
        end
    end
end

```

```

        end
    end
end

```

### TA3.4.4 PEAK\_DETECTION

```

function [y,z] = peak_detection(x, t)
% PEAK_DETECTION maximum positive and negative votage in ERP data
%
% Calculates the maximum positive and negative voltage peaks and their
% latency over the specified time period for each channel. [Y,Z] =
% PEAK_DETECTION(X,T) Where X is the averaged data set and T is the
% time bin over which maximum positive and negative peaks are to be
% calculated. T to be entered as a cell array as time bins may vary. Both
% Y and Z are 3-D cell arrays (i,j,k), where: i = number of channels j =
% 2; where Y{i,1,k} = latency, Y{i,2,k} = voltage k = number of time bins
% Y = maximum positive peaks, Z = maximum negative peaks

no_rows = size(x,1);
no_bins = size(t,2);

max_peak = zeros(no_rows,no_bins);
for i = 1:no_rows;
    for j = 1:no_bins;
        [max_peak(i,j),max_latency_indices(i,j)] = max(x(i,(t{j})));
    end
end

min_peak = zeros(no_rows,no_bins);
for i = 1:no_rows;
    for j = 1:no_bins;
        [min_peak(i,j),min_latency_indices(i,j)] = min(x(i,(t{j})));
    end
end

for i = 1:no_rows;
    for j = 1:no_bins;
        max_latency(i,j) = (t{j}(max_latency_indices(i,j))*4)-100;
    end
end

for i = 1:no_rows;
    for j = 1:no_bins ;
        min_latency(i,j) = (t{j}(min_latency_indices(i,j))*4)-100;
    end
end

y = cell(no_rows,2,no_bins);
for i = 1:no_rows;
    for j = 1:no_bins;
        y{i,1,j} = max_latency(i,j);
        y{i,2,j} = max_peak(i,j);
    end
end

z = cell(no_rows,2,no_bins);
for i = 1:no_rows;
    for j = 1:no_bins;

```

```

        z{i,1,j} = min_latency(i,j);
        z{i,2,j} = min_peak(i,j);
    end
end

```

### TA3.4.5 GRAND\_AVERAGE

**NB** The appropriate AV\_BAD file should be loaded into the workspace and declared a global variable prior to running function.

```

function y = grand_average(x)
% GRAND_AVERAGE averages across subject data
%
% Takes the 3D array X (representing the trial data for all participants
% over 1 condition) and produces the grand average voltage across all
% channels in a double array

global av_bad
no_rows = size(x,1);
no_columns = size(x,2);
no_dim = size(x,3);

grand_total(no_rows,no_columns) = zeros;
for i = 1:no_rows
    for j = 1:no_columns
        for k = 1:no_dim
            if av_bad{k,2}(i,j) == 1
                grand_total(i,j) = grand_total(i,j) + x(i,j,k);
            end
        end
    end
end

no_channels(no_rows,no_columns) = zeros;
for i = 1:no_rows
    for j = 1:no_columns
        for k = 1:no_dim
            no_channels(i,j) = no_channels(i,j) + av_bad{k,2}(i,j);
        end
    end
end

y(no_rows,no_columns) = zeros;
for i = 1:no_rows
    for j = 1:no_columns
        if no_channels(i,:) ~= 0
            y(i,j) = grand_total(i,j)./no_channels(i,j);
        end
    end
end

```

### TA.3.4.6 ROI\_AVERAGE

#### Variations:

Left\_frontal\_average:

Example given below

Left\_central\_average:

ROI reference file: LC = [7 12 13 21 29 30 31 32 36 37 38 42 43 48];

Left\_parietal\_average:

ROI reference file: LP = [51 52 53 54 59 60 61 66 67];

Left\_temporal\_average:

ROI reference file: LT = [35 39 40 41 44 45 46 47 49 50 56 57];

Left\_occipital\_average:

ROI reference file: LO = [58 63 64 65 69 70 71 72 74 75];

Right\_frontal\_average:

ROI reference file: RF = [1 2 3 4 9 10 14 15 122 123 124];

Right\_central\_average:

ROI reference file: RC = [5 81 88 94 99 104 105 106 107 111 112 113 118 119];

Right\_parietal\_average:

ROI reference file: RP = [78 79 80 85 86 87 92 93 98];

Right\_temporal\_average:

ROI reference file: RT = [101 102 103 108 109 110 114 115 116 117 120 121];

Right\_occipital\_average:

ROI reference file: RO = [77 83 84 89 90 91 95 96 97 100];

```
function y = Left_frontal_average(x)
% LEFT_FRONTAL_AVERAGE average voltage across frontal region of interest
%
% Calculates the average voltage in each time sample within the frontal
% region of interest (sensors 18 19 20 23 24 27 28 33 34, as defined by
% Evans & Barrett) of data set X

no_columns = size(x,2);

% region of interest reference file
LF = [18 19 20 22 23 24 25 27 28 33 34];

LF_ref(1:length(LF)) = zeros;
for i = 1:length(LF);
    if sum(x(LF(i),:)) ~= 0;
        LF_ref(i) = 1;
    end
end

LF_total(1:no_columns) = zeros;
for i = 1:no_columns;
    for j = 1:length(LF);
        if LF_ref(j) == 1;
            LF_total(i) = LF_total(i) + x(LF(j),i);
        end
    end
end
```

```

        end
    end
end

av_calc(1,1:no_columns) = sum(LF_ref);

y = LF_total./av_calc;

```

## **TA3.5 Plotting functions**

### **TA3.5.1 FULL\_PLOT**

**NB** Load files to be plotted should be loaded into workspace prior to running script. Example given for plotting the verbal 0-back, verbal 3-back, spatial 0-back, spatial 3-back conditions of a working memory task performed under a depressed mood for one participant.

```

% FULL_PLOT 128 channel plot
%
% Script to display the 128 channels of data in anatomical correct
% positions. Can be used for assessment of raw data, averaged files or
% grand averaged files. Ammend script to include files to be plotted,
% appropriate axis range and file information to axis and legend labels

% CREATING FIGURE CRITERIA AND DIMENSIONS
% Set screensize units to pixels
set (0, 'Units', 'pixels');
% Get screensize
scrnsize = get(0, 'ScreenSize');
% Calculates figure size for 128 channel plot
position = [50 50 0.9*scrnsize(3) 0.9*scrnsize(4)];
% Set figure size
set (figure(1), 'Position', position);
% Sets print/save size to same as figure size
set (gcf, 'PaperPositionMode', 'auto');
% Sets background colour
set (figure(1), 'color', [1 1 1]);
% Sets usable area within figure
set(0,'DefaultAxesPosition', [0.0145 0.0045 0.970 0.950]);
% Set plot layout for 128 channels
plot_layout = [128 0 26 127 22 0 14 126 8 0 125 39 33 27 23 18 17 15 9 ...
2 1 121 44 34 28 24 19 16 10 3 123 122 120 40 35 29 25 20 11 4 124 ...
118 117 116 45 36 30 21 12 6 5 119 112 111 115 41 42 37 31 13 129 113 ...
106 105 104 110 46 47 43 38 7 55 107 88 94 103 109 49 48 52 53 32 62 ...
81 87 93 99 114 50 51 59 60 54 68 80 86 92 98 102 56 58 65 66 61 73 79 ...
85 91 97 108 57 64 70 71 67 76 78 84 90 96 101 63 69 74 75 72 82 77 83 ...
89 95 100];
% Reference file for creation of 128 channel plot
plot_ref = [1 0 1 1 1 0 1 1 1 0 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 ...
1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 ...
1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 ...
1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1];

% Plot graphs

```

```

time = [-100:4:1196];
baseline1 = zeros(1,325);
baseline2 = [-15:0.1:15];

for i = 1:132;
    if plot_ref(i) == 1
        subplot(12,11,i);
        hold on
        plot(time,baseline1, 'color', [0.5 0.5 0.5]);
        plot(time(26),baseline2, 'color', [0.5 0.5 0.5]);
%>> add first file:
        plot(time,s8dv0b_av((plot_layout(i)),:), 'b');
%>> add second file:
        plot(time,s8ds0b_av((plot_layout(i)),:), 'r');
%>> add third file:
        plot(time,s8dv3b_av((plot_layout(i)),:), 'g');
%>> add fourth file:
        plot(time,s8ds3b_av((plot_layout(i)),:), 'm');
        axis([-100 1200 -15 15]);
        axis off;
        set(gca, 'FontSize', 9);
        text(900,9,int2str(plot_layout(i)), 'HorizontalAlignment',...
            'right', 'VerticalAlignment', 'bottom', 'FontSize', 10,...
            'FontWeight', 'bold');
    end
end

% Plot to show scale of channel plots
subplot(12,11,2)
hold on
plot(time,baseline1, 'color', [0.5 0.5 0.5]);
plot(time(26),baseline2, 'color', [0.5 0.5 0.5]);
axis([-100 1200 -15 15]);
set (gca, 'box', 'off');
axis off;
text(-100,0,'|', 'color', [0.5 0.5 0.5], 'Fontsize', 6, 'VerticalAlignment',...
    'middle');
text(100,0,'|', 'color', [0.5 0.5 0.5], 'Fontsize', 6, 'VerticalAlignment',...
    'middle');
text(200,0,'|', 'color', [0.5 0.5 0.5], 'Fontsize', 6, 'VerticalAlignment',...
    'middle');
text(300,0,'|', 'color', [0.5 0.5 0.5], 'Fontsize', 6, 'VerticalAlignment',...
    'middle');
text(400,0,'|', 'color', [0.5 0.5 0.5], 'Fontsize', 6, 'VerticalAlignment',...
    'middle');
text(500,0,'|', 'color', [0.5 0.5 0.5], 'Fontsize', 6, 'VerticalAlignment',...
    'middle');
text(600,0,'|', 'color', [0.5 0.5 0.5], 'Fontsize', 6, 'VerticalAlignment',...
    'middle');
text(700,0,'|', 'color', [0.5 0.5 0.5], 'Fontsize', 6, 'VerticalAlignment',...
    'middle');
text(800,0,'|', 'color', [0.5 0.5 0.5], 'Fontsize', 6, 'VerticalAlignment',...
    'middle');
text(900,0,'|', 'color', [0.5 0.5 0.5], 'Fontsize', 6, 'VerticalAlignment',...
    'middle');
text(1000,0,'|', 'color', [0.5 0.5 0.5], 'Fontsize', 6, 'VerticalAlignment',...
    'middle');
text(1100,0,'|', 'color', [0.5 0.5 0.5], 'Fontsize', 6, 'VerticalAlignment',...

```



```

'middle');
text(1200,0,'(100 ms)','HorizontalAlignment','center',...
'VerticalAlignment','top','FontSize',9);
text(0,-15,'-15','HorizontalAlignment','center',...
'VerticalAlignment','top','FontSize',9);
text(0,15,'15 (microV)','HorizontalAlignment','center', ...
'VerticalAlignment','bottom','FontSize',9);

% Create legend
legend1(1:325) = 8;
legend2(1:325) = 2;
legend3(1:325) = -2;
legend4(1:325) = -8;

subplot(12,11,10)
hold on
plot(time,legend1, 'b');
plot(time,legend2, 'r');
plot(time,legend3, 'g');
plot(time,legend4, 'm');
axis([-100 1200 -10 10]);
axis off
set(gca, 'box', 'off');
text(100,8,'nv0b','HorizontalAlignment','left','VerticalAlignment',...
'bottom','FontSize', 8);
text(-100,2,'ns0b','HorizontalAlignment','left','VerticalAlignment',...
'bottom', 'FontSize', 8);
text(-100,-2,'nv3b','HorizontalAlignment','left','VerticalAlignment',...
'bottom', 'FontSize', 8);
text(-100,-8,'ns3b','HorizontalAlignment','left','VerticalAlignment',...
'bottom', 'FontSize', 8);

clear legend1; clear legend2; clear legend3; clear legend4; clear
plot_layout; clear baseline1; clear baseline2; clear time; clear
plot_ref;
clear scrnsize; clear position; clear i;

```

### TA3.5.2 LOBE\_PLOT\_\*

#### Variations:

lobe\_plot\_p300: to be amended for appropriate source and file information

lobe\_plot\_stroop: to be amended for appropriate source and file information

lobe\_plot\_wm: example given below

```

% LOBE_PLOT_WM Script to plots graphs for the 10 regions of interest
%
% Data plotted for left frontal, right frontal, left central, right
% central, left temporal, right temporal, left parietal, right parietal,
% left occipital and right occipital for working memory tasks.

% Sets screensize units to pixels

```

```

set (0, 'Units', 'pixels');
Gets screensize
scrnsz = get(0, 'ScreenSize');
% Calculates figure size
position = [50 50 0.9*scrnsz(3) 0.9*scrnsz(4)];
% Sets figure size
set (figure(1), 'Position', position);
% Sets print/save size to same as figure size
set (gcf, 'PaperPositionMode', 'auto');
% Sets background colour
set (figure(1), 'color', [1 1 1]);
% Sets usable area within figure
set(0,'DefaultAxesPosition',[0.0145 0.0045 0.970 0.950]);

% Goes to source folder
cd 'Macintosh HD':...

load s1dv0b_rt;load s1dv0b_rp;load s1dv0b_ro;load s1dv0b_rc;
load s1dv0b_rf;load s1dv0b_lt;load s1dv0b_lp;load s1dv0b_lo;
load s1dv0b_lf;load s1dv0b_lc;load s1nv0b_rt;load s1nv0b_rp;
load s1nv0b_ro;load s1nv0b_rc;load s1nv0b_rf;load s1nv0b_lt;
load s1nv0b_lp;load s1nv0b_lo;load s1nv0b_lf;load s1nv0b_lc;
load s1ds0b_rt;load s1ds0b_rp;load s1ds0b_ro;load s1ds0b_rc;
load s1ds0b_rf;load s1ds0b_lt;load s1ds0b_lp;load s1ds0b_lo;
load s1ds0b_lf;load s1ds0b_lc;load s1ns0b_rt;load s1ns0b_rp;
load s1ns0b_ro;load s1ns0b_rc;load s1ns0b_rf;load s1ns0b_lt;
load s1ns0b_lp;load s1ns0b_lo;load s1ns0b_lf;load s1ns0b_lc;
load s1dv3b_rt;load s1dv3b_rp;load s1dv3b_ro;load s1dv3b_rc;
load s1dv3b_rf;load s1dv3b_lt;load s1dv3b_lp;load s1dv3b_lo;
load s1dv3b_lf;load s1dv3b_lc;load s1nv3b_rt;load s1nv3b_rp;
load s1nv3b_ro;load s1nv3b_rc;load s1nv3b_rf;load s1nv3b_lt;
load s1nv3b_lp;load s1nv3b_lo;load s1nv3b_lf;load s1nv3b_lc;
load s1ds3b_rt;load s1ds3b_rp;load s1ds3b_ro;load s1ds3b_rc;
load s1ds3b_rf;load s1ds3b_lt;load s1ds3b_lp;load s1ds3b_lo;
load s1ds3b_lf;load s1ds3b_lc;load s1ns3b_rt;load s1ns3b_rp;
load s1ns3b_ro;load s1ns3b_rc;load s1ns3b_rf;load s1ns3b_lt;
load s1ns3b_lp;load s1ns3b_lo;load s1ns3b_lf;load s1ns3b_lc;

time = [-100:4:1196];
baseline1 = zeros(1,325);
baseline2 = [-12:0.1:12];

subplot(4,4,1)
hold on
h1 = plot(time,baseline1, 'color', [0.5 0.5 0.5]);
h2 = plot(time(26),baseline2, 'color', [0.5 0.5 0.5]);
axis([-100 1200 -12 12]);
set (gca, 'box', 'off');
axis off;

text(-100,0,'-100','HorizontalAlignment','center','VerticalAlignment',...
'top','FontSize',9);
text(100,0,'100','HorizontalAlignment','center','VerticalAlignment',...
'top','FontSize',9);
text(300,0,'300','HorizontalAlignment','center','VerticalAlignment',...
'top','FontSize',9);
text(500,0,'500','HorizontalAlignment','center','VerticalAlignment',...
'top','FontSize',9);

```

```

text(700,0,'700','HorizontalAlignment','center','VerticalAlignment',...
'top','FontSize',9);
text(900,0,'900','HorizontalAlignment','center','VerticalAlignment',...
'top','FontSize',9);
text(1200,0,'1200(ms)','HorizontalAlignment','center',...
'VerticalAlignment','top','FontSize',9);
text(0,-11.9,'- 12 (microV)','HorizontalAlignment','left',...
'VerticalAlignment','top','FontSize',9);
text(0,11.9,'12','HorizontalAlignment','left','VerticalAlignment',...
'bottom','FontSize',9);

subplot(4,4,2)
hold on;
h1 = plot(time,baseline1, 'color', [0.5 0.5 0.5]);
h2 = plot(time(26),baseline2, 'color', [0.5 0.5 0.5]);
h3 = plot(time,sldv0b_lf(:,:),'b-',time,slds0b_lf(:,:),'r-',...
time,sldv3b_lf(:,:),'g-',time,slds3b_lf(:,:),'m-',...
time,slnv0b_lf(:,:),'b--',time,slns0b_lf(:,:),'r--',...
time,slnv3b_lf(:,:),'g--',time,slns3b_lf(:,:),'m--');
axis([-100 1200 -12 12]);
axis off;

set(gca, 'FontSize', 9);
text(-100,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(100,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(200,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(300,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(400,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(500,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(600,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(700,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(800,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(900,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(1000,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(1100,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(1200,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(0,-11.9,'-','HorizontalAlignment','left','VerticalAlignment','top',...
'FontSize',10);
text(0,11.9,'+','HorizontalAlignment','left','VerticalAlignment',...
'bottom','FontSize',10);
title('Left Frontal','color',[0 0 0],'FontSize',10);

subplot(4,4,3)
hold on;

```

```

h1 = plot(time,baseline1, 'color', [0.5 0.5 0.5]);
h2 = plot(time(26),baseline2, 'color', [0.5 0.5 0.5]);
h3 = plot(time,sldv0b_rf(:,:),'b-',time,slds0b_rf(:,:),'r-',...
time,sldv3b_rf(:,:),'g-',time,slds3b_rf(:,:),'m-',...
time,slnv0b_rf(:,:),'b--',time,slns0b_rf(:,:),'r--',...
time,slnv3b_rf(:,:),'g--',time,slns3b_rf(:,:),'m--');
axis([-100 1200 -12 12]);
axis off;
set(gca, 'FontSize', 9);
text(-100,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(100,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(200,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(300,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(400,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(500,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(600,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(700,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(800,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(900,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(1000,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(1100,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(1200,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(0,-11.9,'-','HorizontalAlignment','left','VerticalAlignment','top',...
'FontSize', 10);
text(0,11.9,'+','HorizontalAlignment','left','VerticalAlignment',...
'bottom','FontSize',10);
title('Right Frontal', 'color',[0 0 0],'FontSize',10);

subplot(4,4,6)
hold on;
h1 = plot(time,baseline1,'color',[0.5 0.5 0.5]);
h2 = plot(time(26),baseline2,'color',[0.5 0.5 0.5]);
h3 = plot(time,sldv0b_lc'b-',time,slds0b_lc(:,:),'r-',...
time,sldv3b_lc(:,:),'g-',time,slds3b_lc(:,:),'m-',...
time,slnv0b_lc(:,:),'b--',time,slns0b_lc(:,:),'r--',...
time,slnv3b_lc(:,:),'g--',time,slns3b_lc(:,:),'m--');
axis([-100 1200 -12 12]);
axis off;
set(gca, 'FontSize', 9);
text(-100,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(100,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(200,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(300,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...

```

```

'middle');
text(400,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(500,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(600,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(700,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(800,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(900,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(1000,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(1100,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(1200,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(0,-11.9,'-','HorizontalAlignment','left','VerticalAlignment','top',...
'FontSize',10);
text(0,11.9,'+','HorizontalAlignment','left','VerticalAlignment',...
'bottom','FontSize',10);
title('Left Central','color',[0 0 0],'FontSize',10);

subplot(4,4,7)
hold on;
h1 = plot(time,baseline1, 'color', [0.5 0.5 0.5]);
h2 = plot(time(26),baseline2, 'color', [0.5 0.5 0.5]);
h3 = plot(time,sldv0b_rc(:,:),'b-',time,slds0b_rc(:,:),'r-',...
time,sldv3b_rc(:,:),'g-',time,slds3b_rc(:,:),'m-',...
time,slnv0b_rc(:,:),'b--',time,slns0b_rc(:,:),'r--',...
time,slnv3b_rc(:,:),'g--',time,slns3b_rc(:,:),'m--');
axis([-100 1200 -12 12]);
axis off;
set(gca, 'FontSize', 9);
text(-100,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(100,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(200,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(300,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(400,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(500,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(600,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(700,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(800,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(900,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(1000,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(1100,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...

```

```

'middle');
text(1200,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(0,-11.9,'-','HorizontalAlignment','left','VerticalAlignment','top',...
'FontSize',10);
text(0,11.9,'+','HorizontalAlignment','left','VerticalAlignment',
'bottom','FontSize',10);
title('Right Central','color',[0 0 0],'FontSize',10);

subplot(4,4,9)
hold on;
h1 = plot(time,baseline1, 'color', [0.5 0.5 0.5]);
h2 = plot(time(26),baseline2, 'color', [0.5 0.5 0.5]);
h3 = plot(time,sldv0b_lt(:,:),'b-',time,slds0b_lt(:,:),'r-',...
time,sldv3b_lt(:,:),'g-',time,slds3b_lt(:,:),'m-',...
time,slnv0b_lt(:,:),'b--',time,slns0b_lt(:,:),'r--',...
time,slnv3b_lt(:,:),'g--',time,slns3b_lt(:,:),'m--');
axis([-100 1200 -12 12]);
axis off;
set(gca, 'FontSize', 9);
text(-100,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(100,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(200,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(300,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(400,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(500,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(600,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(700,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(800,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(900,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(1000,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(1100,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(1200,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(0,-11.9,'-','HorizontalAlignment','left','VerticalAlignment','top',...
'FontSize',10);
text(0,11.9,'+','HorizontalAlignment','left','VerticalAlignment',...
'bottom','FontSize',10);
title('Left Temporal','color',[0 0 0],'FontSize',10);

subplot(4,4,10)
hold on;
h1 = plot(time,baseline1,'color',[0.5 0.5 0.5]);
h2 = plot(time(26),baseline2, 'color', [0.5 0.5 0.5]);
h3 = plot(time,sldv0b_lp(:,:),'b-',time,slds0b_lp(:,:),'r-',...
time,sldv3b_lp(:,:),'g-',time,slds3b_lp(:,:),'m-',...
time,slnv0b_lp(:,:),'b--',time,slns0b_lp(:,:),'r--',...

```

```

time,slnv3b_lp(:,:),'g--',time,slns3b_lp(:,:),'m--');
axis([-100 1200 -12 12]);
axis off;
set(gca, 'FontSize', 9);
text(-100,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(100,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(200,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(300,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(400,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(500,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(600,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(700,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(800,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(900,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(1000,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(1100,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(1200,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(0,-11.9,'-', 'HorizontalAlignment','left','VerticalAlignment','top',...
'FontSize',10);
text(0,11.9,'+', 'HorizontalAlignment','left','VerticalAlignment',...
'bottom','FontSize',10);
title('Left Parietal','color',[0 0 0],'FontSize',10);

subplot(4,4,11)
hold on;
h1 = plot(time,baseline1, 'color', [0.5 0.5 0.5]);
h2 = plot(time(26),baseline2, 'color', [0.5 0.5 0.5]);
h3 = plot(time,sldv0b_rp(:,:),'b-',time,slds0b_rp(:,:),'r-',...
time,sldv3b_rp(:,:),'g-',time,slds3b_rp(:,:),'m-',...
time,slnv0b_rp(:,:),'b--',time,slns0b_rp(:,:),'r--',...
time,slnv3b_rp(:,:),'g--',time,slns3b_rp(:,:),'m--');
axis([-100 1200 -12 12]);
axis off;
set(gca, 'FontSize', 9);
text(-100,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(100,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(200,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(300,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(400,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(500,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');

```

```

text(600,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(700,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(800,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(900,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(1000,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(1100,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(1200,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(0,-11.9,'-','HorizontalAlignment','left','VerticalAlignment','top',...
'FontSize',10);
text(0,11.9,'+','HorizontalAlignment','left','VerticalAlignment',...
'bottom','FontSize',10);
title('Right Parietal','color',[0 0 0],'FontSize', 10);

subplot(4,4,12)
hold on;
h1 = plot(time,baseline1, 'color', [0.5 0.5 0.5]);
h2 = plot(time(26),baseline2, 'color', [0.5 0.5 0.5]);
h3 = plot(time,sldv0b_rt(:,:),'b-',time,slds0b_rt(:,:),'r-',...
time,sldv3b_rt(:,:),'g-',time,slds3b_rt(:,:),'m-',...
time,slnv0b_rt(:,:),'b--',time,slns0b_rt(:,:),'r--',...
time,slnv3b_rt(:,:),'g--',time,slns3b_rt(:,:),'m--');
axis([-100 1200 -12 12]);
axis off;
set(gca, 'FontSize', 9);
text(-100,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(100,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(200,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(300,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(400,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(500,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(600,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(700,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(800,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(900,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(1000,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(1100,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(1200,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(0,-11.9,'-','HorizontalAlignment','left','VerticalAlignment','top',...

```



```

'FontSize',10);
text(0,11.9,'+', 'HorizontalAlignment','left','VerticalAlignment',...
'bottom','FontSize',10);
title('Right Temporal','color',[0 0 0],'FontSize',10);

subplot(4,4,14)
hold on;
h1 = plot(time,baseline1, 'color', [0.5 0.5 0.5]);
h2 = plot(time(26),baseline2, 'color', [0.5 0.5 0.5]);
h3 = plot(time,sldv0b_lo(:,:), 'b-',time,slds0b_lo(:,:), 'r-',...
time,sldv3b_lo(:,:), 'g-',time,slds3b_lo(:,:), 'm-',...
time,slnv0b_lo(:,:), 'b--',time,slns0b_lo(:,:), 'r--',...
time,slnv3b_lo(:,:), 'g--',time,slns3b_lo(:,:), 'm--');
axis([-100 1200 -12 12]);
axis off;
set(gca, 'FontSize', 9);
text(-100,0,'|', 'color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(100,0,'|', 'color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(200,0,'|', 'color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(300,0,'|', 'color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(400,0,'|', 'color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(500,0,'|', 'color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(600,0,'|', 'color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(700,0,'|', 'color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(800,0,'|', 'color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(900,0,'|', 'color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(1000,0,'|', 'color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(1100,0,'|', 'color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(1200,0,'|', 'color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(0,-11.7,'-', 'HorizontalAlignment','left','VerticalAlignment','top',...
'FontSize',10);
text(0,11.9,'+', 'HorizontalAlignment','left','VerticalAlignment',...
'bottom','FontSize',10);
title('Left Occipital','color',[0 0 0],'FontSize',10);

subplot(4,4,15)
hold on;
h1 = plot(time,baseline1, 'color', [0.5 0.5 0.5]);
h2 = plot(time(26),baseline2, 'color', [0.5 0.5 0.5]);
h3 = plot(time,sldv0b_ro(:,:), 'b-',time,slds0b_ro(:,:), 'r-',...
time,sldv3b_ro(:,:), 'g-',time,slds3b_ro(:,:), 'm-',...
time,slnv0b_ro(:,:), 'b--',time,slns0b_ro(:,:), 'r--',...
time,slnv3b_ro(:,:), 'g--',time,slns3b_ro(:,:), 'm--');

```

```

axis([-100 1200 -12 12]);
axis off;
set(gca, 'FontSize', 9);
text(-100,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(100,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(200,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(300,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(400,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(500,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(600,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(700,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(800,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(900,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(1000,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(1100,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(1200,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(0,-11.7,'-', 'HorizontalAlignment','left','VerticalAlignment','top',...
'FontSize',10);
text(0,11.9,'+', 'HorizontalAlignment','left','VerticalAlignment',...
'bottom','FontSize',10);
title('Right Occipital','color',[0 0 0],'FontSize',10);

% Plots legend for data.
h4 = legend(h3,'depressed v0b','depressed s0b','depressed v3b','depressed
s3b', 'neutral v0b', 'neutral s0b', 'neutral v3b', 'neutral s3b');

% Set legend font size (Code by S.Rievers (1999))
hc = get(h4, 'Children');
for i = 1:length(hc)
    if strcmp(get(hc(i),'Type'),'text')
        set (hc(i),'FontUnits','points');
        set (hc(i),'FontSize',10);
    end;
end;

set(h4,'visible','off');
legend_position = [0.850 0.800 0.0986 0.1484];
set(legend, 'position', legend_position);

clear sldv0b_rt;clear sldv0b_rp;clear sldv0b_ro;clear sldv0b_rc;
clear sldv0b_rf;clear sldv0b_lt;clear sldv0b_lp;clear sldv0b_lo;
clear sldv0b_lf;clear sldv0b_lc;clear slnv0b_rt;clear slnv0b_rp;
clear slnv0b_ro;clear slnv0b_rc;clear slnv0b_rf;clear slnv0b_lt;
clear slnv0b_lp;clear slnv0b_lo;clear slnv0b_lf;clear slnv0b_lc;
clear slds0b_rt;clear slds0b_rp;clear slds0b_ro;clear slds0b_rc;

```

```

clear slds0b_rf;clear slds0b_lt;clear slds0b_lp;clear slds0b_lo;
clear slds0b_lf;clear slds0b_lc;clear slns0b_rt;clear slns0b_rp;
clear slns0b_ro;clear slns0b_rc;clear slns0b_rf;clear slns0b_lt;
clear slns0b_lp;clear slns0b_lo;clear slns0b_lf;clear slns0b_lc;
clear sldv3b_rt;clear sldv3b_rp;clear sldv3b_ro;clear sldv3b_rc;
clear sldv3b_rf;clear sldv3b_lt;clear sldv3b_lp;clear sldv3b_lo;
clear sldv3b_lf;clear sldv3b_lc;clear slnv3b_rt;clear slnv3b_rp;
clear slnv3b_ro;clear slnv3b_rc;clear slnv3b_rf;clear slnv3b_lt;
clear slnv3b_lp;clear slnv3b_lo;clear slnv3b_lf;clear slnv3b_lc;
clear slds3b_rt;clear slds3b_rp;clear slds3b_ro;clear slds3b_rc;
clear slds3b_rf;clear slds3b_lt;clear slds3b_lp;clear slds3b_lo;
clear slds3b_lf;clear slds3b_lc;clear slns3b_rt;clear slns3b_rp;
clear slns3b_ro;clear slns3b_rc;clear slns3b_rf;clear slns3b_lt;
clear slns3b_lp;clear slns3b_lo;clear slns3b_lf;clear slns3b_lc;
clear time;clear baseline1;clear baseline2;clear position;
clear scrnsize;clear h1;clear h2;clear h3;clear h4;clear hc;
clear legend_position;

```

### TA3.5.3 CHANNEL\_PLOT

**NB** To be amended for appropriate source and file information. This example is for the right frontal regions of interest, with a plot of oddball target and standard data recorded under neutral and depressed moods

```

% CHANNEL_PLOT Plots a single comparison graph, can be used for
% displaying single channel data or grand averages across regions of
% interest

position = [256 334 600 450];
set (figure(1), 'Position', position);
set (gcf, 'PaperPositionMode', 'auto');

% Source data - ammend as appropriate
cd 'Macintosh HD':...
load dstanrf; load dtarrf; load nstanrf; load ntarrf;

set (figure(1), 'color', [1 1 1]);
set(0,'DefaultAxesPosition', [0.0500 0.0200 0.900 0.930]);

time = [-100:4:696];
baseline1 = zeros(1,200);
baseline2 = [-10:0.01:10];
hold on;
h1 = plot(time,baseline1, 'color', [0.5 0.5 0.5]);
h2 = plot(time(26),baseline2, 'color', [0.5 0.5 0.5]);
h3 = plot(time,dstanrf(:,:),'b',time,dtarrf(:,:),'r',...
time,nstanrf(:,:),'g',time,ntarrf(:,:),'m');
axis([-100 700 -10 10]);
axis off;
set(gca, 'FontSize', 9);
text(100,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(200,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(300,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...

```

```

'middle');
text(400,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(500,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(600,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(700,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
%text(800,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
%text(900,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
%text(1000,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
%text(1100,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
%text(1200,0,'|','color',[0.5 0.5 0.5],'FontSize',6,'VerticalAlignment',...
'middle');
text(-100,0,'-100','HorizontalAlignment','right','VerticalAlignment',...
'top','FontSize',9);
text(100,0,'100','HorizontalAlignment','center','VerticalAlignment',...
'top','FontSize',9);
text(300,0,'300','HorizontalAlignment','center','VerticalAlignment',...
'top','FontSize',9);
text(500,0,'500','HorizontalAlignment','center','VerticalAlignment',...
'top','FontSize',9);
%text(700,0,'700','HorizontalAlignment','center','VerticalAlignment',...
'top','FontSize',9);
%text(900,0,'900','HorizontalAlignment','center','VerticalAlignment',...
'top','FontSize',9);
%text(1100,0,'1100','HorizontalAlignment','center','VerticalAlignment',...
'top','FontSize',9);
text(700,0,'700(ms)','HorizontalAlignment','center','VerticalAlignment',...
'top','FontSize',9);
text(0,-9.9,'-10
(microV)','HorizontalAlignment','left','VerticalAlignment',...
'top','FontSize',9);
text(0,9.9,'10','HorizontalAlignment','left','VerticalAlignment',...
'bottom','FontSize',9);
title('right frontal ROI','color',[0 0 0],'FontSize',12);
h4 = legend(h3,'depressed standard','depressed target', 'neutral
standard', 'neutral target', 0);

% Set legend font size (% Code by S.Rievers (1999))
hc = get(h4, 'Children');
for i = 1:length(hc)
    if strcmp(get(hc(i),'Type'),'text')
        set(hc(i),'FontUnits','points');
        set(hc(i),'FontSize',10);
    end;
end;

set(h4,'visible','off');

```

---

## **Technical Appendix 4**

### **EPRIME/EBANS routine for a 3-back working memory task**

---

#### **TA4.1 Introduction**

There were a number of basic experimental paradigms provided with the E-PRIME stimulus presentations software which could be adapted for the oddball and Stroop tasks required. However, none were similar to the more complex design of the 3-back working memory task and therefore a novel routine had to be created.

#### **TA4.2 Design specifications**

There are three independent variables: mood (neutral, depressed), memory workload (0-back, 3-back) and modality (spatial, verbal). Within the depressed and neutral sessions, the participant will complete a mood induction procedure, either side of which they will complete a mood assessment. The participant will then complete a 0-back working memory task. They will be presented with a fixation (●) for 500 ms. A stimulus display will then present a letter (b, c, d, g, j, or k), either in upper- or lower-case in one of six positions on the screen, for 250 ms. The background display will remain on the screen for 2000 ms while the participant makes their response. During the inter-trial interval of 500 ms, the background display will remain on screen.

The participant will complete a spatial and verbal version of the 0-back task. They will be asked to press '1' if the stimuli they are told to compare are the same and '2' if they are different. Next they will complete a training session for the 3-back task. The same stimulus presentation routine will be used, only the instructions will differ. The participants will be told that they must compare each stimuli with that they saw three trials previously. Again there will be a spatial and verbal version of the task and responses made with the keys '1' and '2'.

The participant will then complete 4 blocks of the 3-back task, 2 verbal and 2 spatial. Prior to each block the participant will be presented with 5 mood statements from the series presented earlier in the session. Finally, the participant will complete a third mood assessment form.

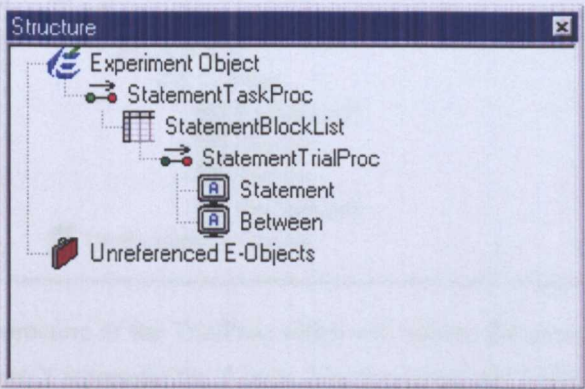
### **TA4.3 Stimulus Presentation**

There are two stimulus presentation procedures which will be used in this experiment: **StatementTrialProc** will control the presentation of the mood induction statements and a **TrialProc** will control the presentation of the 0-back task, 3-back training and 3-back task conditions.

#### **TA4.3.1 StatementTrialProc**

The **StatementTrialProc** trial will consist of two events: statement and blank, and will be controlled by the **StatementTaskProc** (Figure TA4.1). The properties

assigned to the events within the **StatementTrialProc** events are summarised in Table TA4.1.



**Figure TA4.1** The basic structure of the **StatementTaskProc** which will control the mood induction section of the experiment.

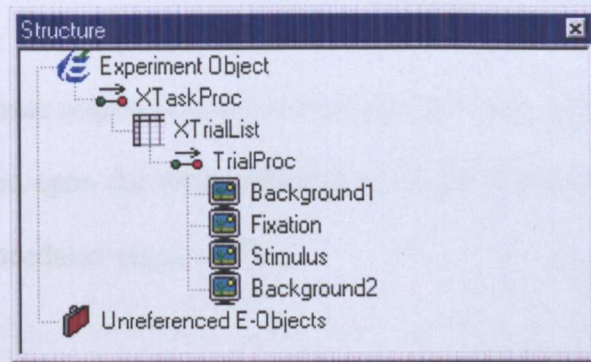
Object	Fixed Properties	Varying Properties
Statement	<b>Text Display Object</b>	
	• Justification: centre	
	• Duration: 10000 ms	• [stimulus] (statement 1, statement 2, ...)
	• Fore colour: black	
	• Background colour: white	
Blank	• Font: Garamond, 30pt	
	<b>Text Display Object</b>	
	• Duration :1000 ms	• none
	• Background colour: white	

**Table TA4.1** The fixed and varying properties of the objects within the **StatementTrialProc**.

**TA4.3.2 TrialProc**

The stimulus presentation procedure will be the same in the 0-back, 3-back training and 3-back task conditions. The **TrialProc** will consist of four events: Background1, Fixation, Stimulus and Background2. This procedure will be controlled by **XTaskProc**, where X will indicate the control (Control), 3-back training (Training) or 3-back task (NBack) condition (Figure TA2.2). The properties assigned to these events are summarised in Table TA4.2.





**Figure TA4.2** The basic structure of the TrialProc which will control the stimulus presentation under the XTaskProc procedure, where X represents the 0-back, 3-back training and 3-back task conditions.

Object	Fixed Properties	Varying Properties
Background1	<b>Image Display Object</b> <ul style="list-style-type: none"> <li>• Bitmap: montage of letters</li> <li>• Presentation: centre screen</li> <li>• Duration: 500 ms</li> </ul>	<ul style="list-style-type: none"> <li>• None</li> </ul>
Fixation	<b>Image Display Object</b> <ul style="list-style-type: none"> <li>• Bitmap: montage of letters with a central fixation point</li> <li>• Presentation:centre screen</li> <li>• Duration: 500 ms</li> </ul>	<ul style="list-style-type: none"> <li>• None</li> </ul>
Stimulus	<b>Image Display Object</b> <ul style="list-style-type: none"> <li>• Bitmap: [stimulus]</li> <li>• Presentation: centre screen</li> <li>• Duration: 250 ms</li> </ul>	<ul style="list-style-type: none"> <li>• [Stimulus] – one of 72 stimulus bitmaps (the letters b, c, d, g, j and k in upper and lower case in six different positions, presented on the montage of letters)</li> </ul>
Background2	<b>Image Display Object</b> <ul style="list-style-type: none"> <li>• Bitmap: montage of letters</li> <li>• Presentation: centre screen</li> <li>• Duration: 2000 ms</li> <li>• Allowable input: '1' and '2'</li> </ul>	<ul style="list-style-type: none"> <li>• Response Collection – when two stimuli are the same the correct response is '1' and when two stimuli are different the correct response is '2'</li> </ul>

**Table TA4.2** The fixed and varying properties of the objects within the TrialProc.

#### **TA4.4 Experimental session structure**

Each experimental session will contain 4 different task procedures:

- **StatementTaskProc:** responsible for controlling the statement presentation during the mood induction procedure;



- **ControlTaskProc**: responsible for controlling the 0-back task;
- **TrainingTaskProc**: responsible for controlling the 3-back task training component;
- **NBackTaskProc**: responsible for controlling the 3-back task.

The **TaskList**, situated upon the **SessionProc** level of the hierarchy, will prompt E-Prime to run these procedures sequentially.

#### **TA4.4.1 Session Procedure**

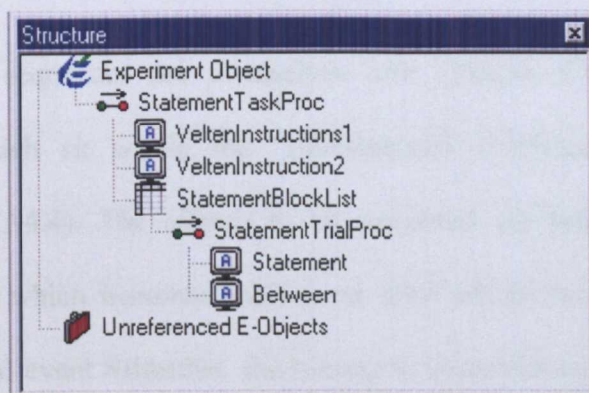
The participant paces their progression through the experiment. Once the verbal instructions have been given to the participant, the experimenter will leave the cubicle. A *Welcome* Text Object will display the following text: “*Welcome to the experiment* Press 1 to continue”

The participant is now required to fill out the first of the mood assessment form, which are included in their ‘Subject Booklet’. The Text Object *MA1* instructs them to: “Please fill out form 1 in your subject booklet – answer the questions as quickly and accurately as possible Press 1 when you have completed the form”. Upon completion, the Text Object *ThankYou1* will be displayed: “Thank You Press 1 to continue”

The four task procedures will then be run in sequence, after which **MA3** and **ThankYou3** and will start and finish the final mood assessment. **GoodBye** will then thank the participant for taking part: “Thank you very much for participating Please remain seated until the experimenter enters the cubicle”

#### TA4.4.1.1 StatementTaskProc

The structure of the **StatementTaskProc** is shown in Figure TA4.3. The instructions for the induction procedure will be in an 'Instruction Booklet'. The participants are prompted to read this by the Text Object **VeltenInstructions1**: "Please read through the Instruction Booklet When you have completed the booklet – press 1 to continue". The presentation of the statements are initiated by **VeltenInstructions2**: "The next slide will begin the series of statements – remember to read each statement to yourself and then out loud Press 1 to continue".



**Figure TA4.3** The structure of the **StatementTaskProc** that will control the presentation of the Velten mood induction procedure

#### TA4.4.1.2 ControlTaskProc

This procedure contains two blocks, a spatial and verbal version of the task. By using nested lists, the trial procedure selects different stimulus sequences for each block while using the same overall procedure. To do this a List Object **ControlBlockList** is required which outlines the blocks to be used [ControlType] and the procedures they will follow [Procedure].

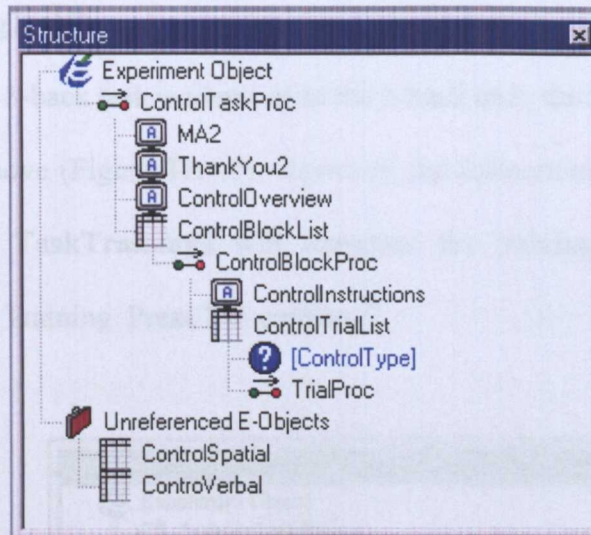
Within the **ControlBlockProc**, a second List Object **ControlTrialList** detail what will happen during each block at the trial level (Table TA4.3). Here, the attribute [ControlType] is referenced as a ‘nested’ attribute. This allows each block to choose a different sequence of presentation, according to value of the **ControlType** attribute in the **ControlBlockList**.

<i>Weight</i>	<b>Nested</b>	<b>Procedure</b>	<b>Stimulus</b>	<b>CorrectAnswer</b>
1	[ControlType]	TrialProc	[ListStim]	[CorrectAnswer]

**Table TA4.3** The **ControlTrialList** which details the trial level procedure within each block.

These presentation sequences are themselves List Objects, **ControlSpatial** and **ControlVerbal**, which sit within the ‘Unreferenced E-Objects’ branch of the hierarchy (Figure TA4.4). The stimuli to be presented are referenced under the attribute [ListStim], which becomes the lowest level of the stimulus presentation hierarchy. In the trial event **Stimulus**, the bitmap to be presented is declared as the attribute [Stimulus]. When E-Prime references this attribute within the **ControlTrialList**, it sees that the attribute [ListStim] is referenced, which is contained within a nested list referenced by the attribute [ControlType]. Therefore, E-Prime will go to the appropriate nested list (**ControlSpatial** or **ControlVerbal**) and present the bitmaps listed under the attribute [ListStim]. As the correct answer will vary depending on the list of stimuli presented, the attribute [CorrectAnswer] is referenced in the same way.



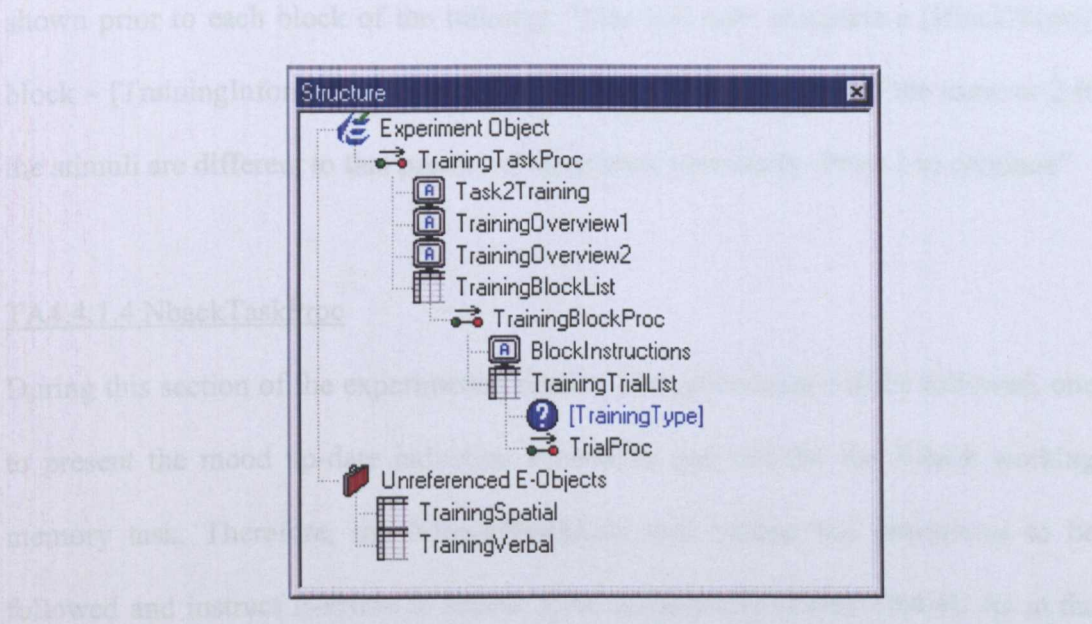


**Figure TA4.4** The structure of the ControlTaskProc that will control the presentation of the 0-back working memory task.

As the instructions to the participant will only differ in terms of whether the trial is verbal or spatial, the attributes [BlockName] and [ControlInformation] were added to the **ControlBlockList**. Then, through the Text Object **ControlInstructions**, these attributes can be referenced in order to make the instructions modality specific: “You will now complete a [BlockName] block – [ControlInformation] Press 1 to continue”. For the spatial block the instruction ‘therefore you will be focusing on the position of the stimulus’ will be used and for the verbal block the instruction ‘therefore you will be focusing on the letter presented’ will be used.

TA4.4.1.3 TrainingTaskProc

As the basis of the 3-back task is identical to the 0-back task, the overall structure will be as described above (Figure TA4.5). However, the instruction-based Text Objects will be different. **TaskTraining2** will introduce the training component of the session: “Task 2 – Training Press 1 to continue”



**Figure TA4.5** The structure of the TrainingTaskProc that will control the presentation of the 3-back training section.

To remind the participants of the aim of the 3-back task, **TrainingOverview1** will present the following information: “You will be presented with a series of stimuli and your task is to compare each stimulus with the stimulus you saw three trials previously In the verbal task you will be comparing the stimuli on the basis of the letter presented In the spatial task you will be comparing the stimuli on the basis of the position of presentation Press 1 to continue”.



Next, to remind the participant of the responses they must make, TrainingOverview2 is presented: “If a stimulus is the same as that three trials previously – press 1 If a stimulus is different to that three trials previously – press 2”.

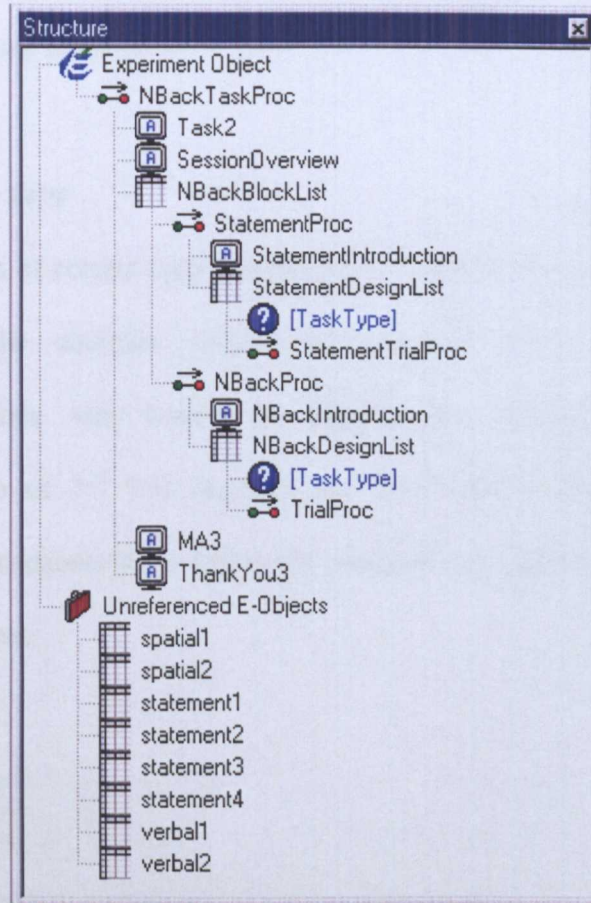
Using the attribute referencing procedure described above, **BlockInstructions** will be shown prior to each block of the training: “You will now complete a [BlockName] block – [TrainingInformation] Remember = press 1 if the stimuli are the same or 2 if the stimuli are different to that presented three trials previously Press 1 to continue”

#### TA4.4.1.4 NbackTaskProc

During this section of the experimental session two procedures will be followed, one to present the mood up-date induction statements and one for the 3-back working memory task. Therefore, the **NBackBlockList** will outline the procedures to be followed and instruct E-Prime to follow them sequentially (Table TA4.4). As in the previous two examples, the nested list procedure will be used to present all stimuli (Figure TA4.6).

<i>Weight</i>	<b>Nested</b>	<b>Procedure</b>	<b>TaskType</b>	<b>BlockName</b>
1		StatementProc	statement1	statements
1		NBackProc	spatial1	spatial
1		StatementProc	statement2	statements
1		NBackProc	verbal1	verbal
1		StatementProc	statement3	statements
1		NBackProc	spatial2	spatial
1		StatementProc	statement4	statements
1		NBackProc	verbal4	verbal

**Table TA4.4** The NBackBlockList used to control the sequence of events during the NBackTaskBlock.



**Figure TA4.6** The NBackTaskProc which will run the alternating statement presentation and 3-back task section of the session.

**Task2** will introduce this component of the session: “You will now complete four blocks of the 3-back task (2 spatial and 2 verbal) Before each block you will be presented with 5 statements from the series presented earlier – remember to read each statement to yourself and out loud Press 1 to continue”. Prior to the presentation of statements, **StatementIntro** will be presented: “You will now be presented with the [BlockName] Press 1 to continue”. While the 3-back task will be preceded by **NBackIntro**: “You will now complete a [BlockName] block Press 1 to continue”.

Both these Objects will use the [BlockName] attribute within the [NBackBlockList] to insert the appropriate block name.

#### *TA4.4.1.4.1 Stimulus Lists*

For the classification of correct response during the 3-back task, each stimulus must be compared to the stimulus which was presented three trials previously. Unfortunately E-Prime was unable to support this method of comparison. Furthermore, a ratio of 3:7 was required for the 'same'/'different' comparisons between stimuli. Consequently the following program was used to compile possible presentation sequences:

```
#include <iostream>
#include <valarray>
#include <fstream>

using namespace std; //introduces namespace std

void random_valarray(valarray<int>* p);
void print_valarray(valarray<int>* p);
int check_valarray(valarray<int>* p);

ofstream out_file("output.txt");

const int n_back=3;
const int no_of_stims=6;
const int no_of_stim_types=12;
const int letter_hits_lower=18;
const int letter_hits_upper=72;
const int pos_hits_lower=18;
const int pos_hits_upper=72;

int main()
{
    out_file<<"sequences" << endl;
    srand(time(NULL));

    for(int i=0; i<10000000; i++)
    {
        valarray<int> trials(-
1,no_of_stims*no_of_stim_types);
        random_valarray(&trials);
        if(check_valarray(&trials))
            print_valarray(&trials);
    }
}
```



```

        out_file<<"Done";
        return 0;
    }

void random_valarray(valarray<int>* p)
{
    for(int i=0; i<(*p).size(); i++)
        (*p)[i]=i;

    for(int i=0; i<(*p).size()*(*p).size(); i++)
    {
        int a=rand()%(*p).size();
        int b=rand()%(*p).size();
        int temp=(*p)[a];
        (*p)[a]=(*p)[b];
        (*p)[b]=temp;
    }
}

void print_valarray(valarray<int>* p)
{
    for(int i=0; i<(*p).size(); i++)
        out_file<<(*p)[i]<<"\t";
    out_file<<endl;
}

int check_valarray(valarray<int>* p)
{
    int letter_hits=0;

    for(int i=n_back; i<(*p).size(); i++)
        if((*p)[i-n_back]/no_of_stim_types==(*p)[i]/no_of_stim_types)
            letter_hits++;

    if(letter_hits<letter_hits_lower ||
letter_hits>letter_hits_upper)
        return 0;

    int pos_hits=0;

    for(int i=n_back; i<(*p).size(); i++)
        if((( *p)[i-n_back]%no_of_stim_types)/2==(( *p)[i]%no_of_stim_types)/2)
            pos_hits++;

    if( pos_hits<pos_hits_lower || pos_hits>pos_hits_upper )
        return 0;

    out_file<<pos_hits<<"\t"<<letter_hits<<"\t";

    return 1;
}

```

The generated lists were then transferred to an Excel spreadsheet in order to test the distribution of verbal and spatial correct responses. A suitable list would contain a pseudo-random distribution of hits (spatial and verbal) with the following criteria:

- there should be 19 correct spatial and 19 correct verbal hits within the list;
- there should be no more than three correct hits in a row;
- each hit should be a correct response for a spatial OR verbal trial.

Sixty-four lists were chosen so that each 3-back task was associated with a different sequence of stimuli. Individual [StimulusLists] were created for experimental sessions which also contained the appropriate correct responses. Due to the constrained distribution of the stimuli within each list, those not used in the N-back task were used for training and the 0-back task.

#### **TA4.5 Adding the EBANS PackageCall objects**

E-Prime calls NetStation using EBANS PackageCall Objects. The first PackageCall sets up the communication link between the computers controlling E-Prime and NetStation. The routine used to do this is *Init* and therefore, the object is named *NSInit*.

Within the routine *Init*, the user must set the parameter *objCellMap*. This parameter refers to the List Object that details the cells (conditions) of the experiment. Within this study there are three List Objects which will contain information on the cells of the experiment: **ControlTrialList**, **TrainingTrialList** and **NBackTrialList**. As the

routine *Init* can only access one List Object, a *CellList* must be created that contains all the cell information for the session (Table TA4.5).

<i>Weight</i>	<b>Nested</b>	<b>Procedure</b>	<b>CellNumber</b>	<b>CellLabel</b>
			1	SpatialControl
			2	VerbalControl
			3	SpatialTraining
			4	VerbalTraining
			5	SpatialTask
			6	VerbalTask

**Table TA4.5** The CellList for the working memory task which contains condition information for the 0-back, training and 3-back tasks.

As the stimuli associated with the cells are in nested lists, they will contain information on the appropriate CellNumber and CellLabel for each stimulus presented, which can be called by the above CellList.

E-Prime is also able to send stimulus information to the NetStation recording that can be used during the segmentation of the EEG data. Therefore a **KeyList** was created that contains information on the stimulus presented, the condition under which it was presented and the induced mood.

E-Prime also send NetStation a tag every time a an event in a trial is presented. For all three ERP recording conditions the same trial structure is used: *Background1*,

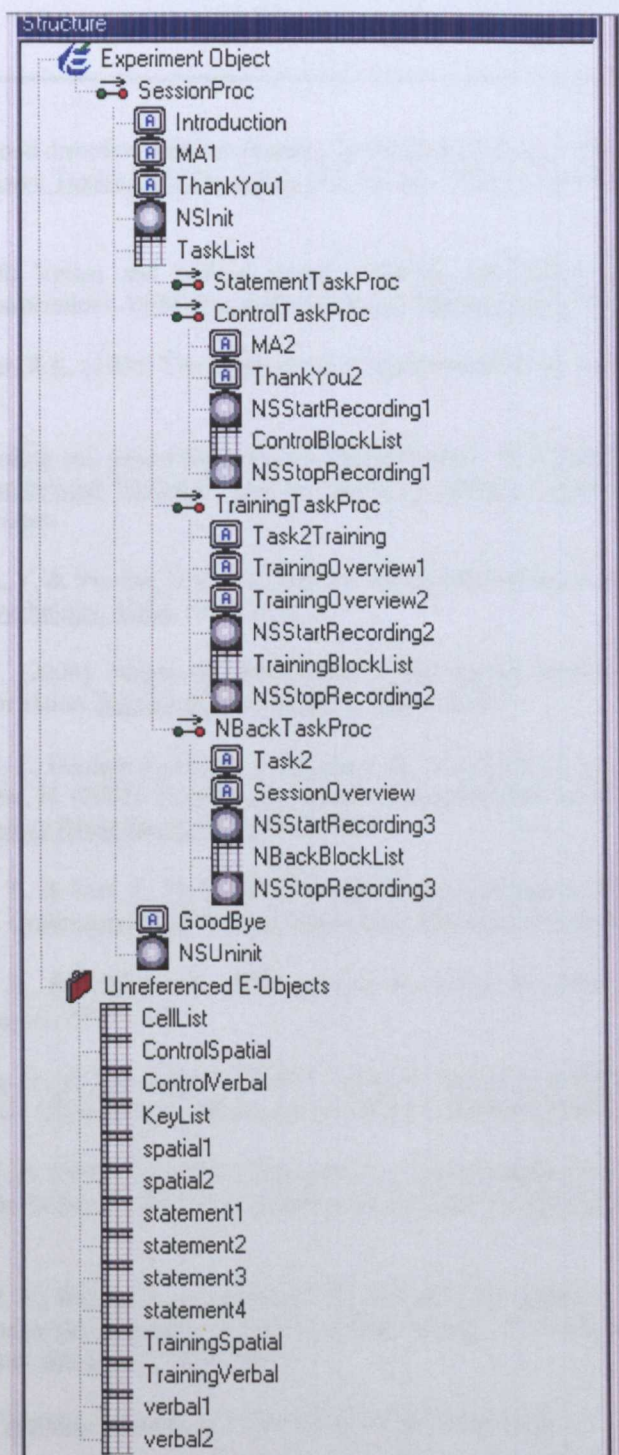
*Fixation*, *Stimulus* and *Background2*. The tags for the first three events will be identical, however the tag associated with *Background2* must differentiate between the conditions, as it is here that the participant response is made and recorded. Therefore the script within *NSTrialEvent* must be written so that the appropriate tag is used depending on the *CellLabel* of the stimulus:

```
NetStation_SendTrialEvent c, Background1, "pre+"
NetStation_sendTrialEvent c, Fixation, "fix+"
NetStation_SendTrialEvent c, Stimulus, "stim"
```

```
Selecr Case c.GetAttrib ("CellLabel")
    Case "SpatialControl"
        Background2.Tag = "scon"
    Case "VerbalControl"
        Background2.Tag = "vcon"
    Case "SpatialTraining"
        Background2.Tag = "strn"
    Case "VerbalTraining"
        Background2.Tag = "vtrn"
    Case "SpatialTask"
        Background2.Tag = "stsk"
    Case "VerbalTraining"
        Background2.Tag = "vtsk"
```

The full structure of a neutral mood session is shown in Figure TA4.7. As the depressed mood condition requires the reversal of the mood induction at the end of the experimental session, a **ReverseTaskProc** is added to the end of the experiment for the presentation of the relation Velten statements.

## References



**Figure TA4.7** The full structure of the neutral experimental session, including all NetStation PackageCall.

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